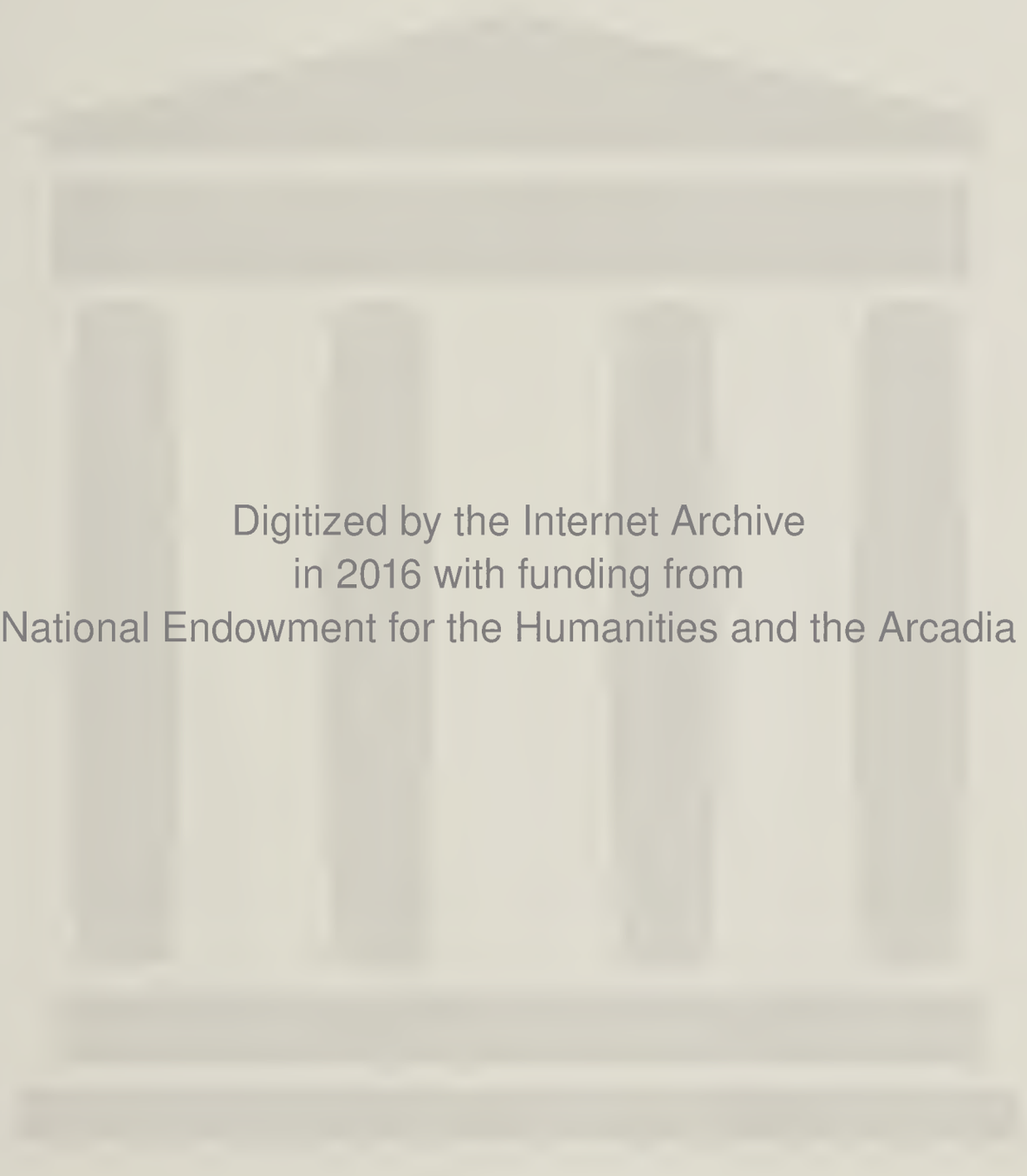
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JOURNAL OF ARIZONA MEDICAL ASSOCIATION

MEDICAL SOCIETY OF THE UNITED STATES AND MEXICO

MINI SYMPOSIUM
American College of Physicians
Review of Arthritic Conditions

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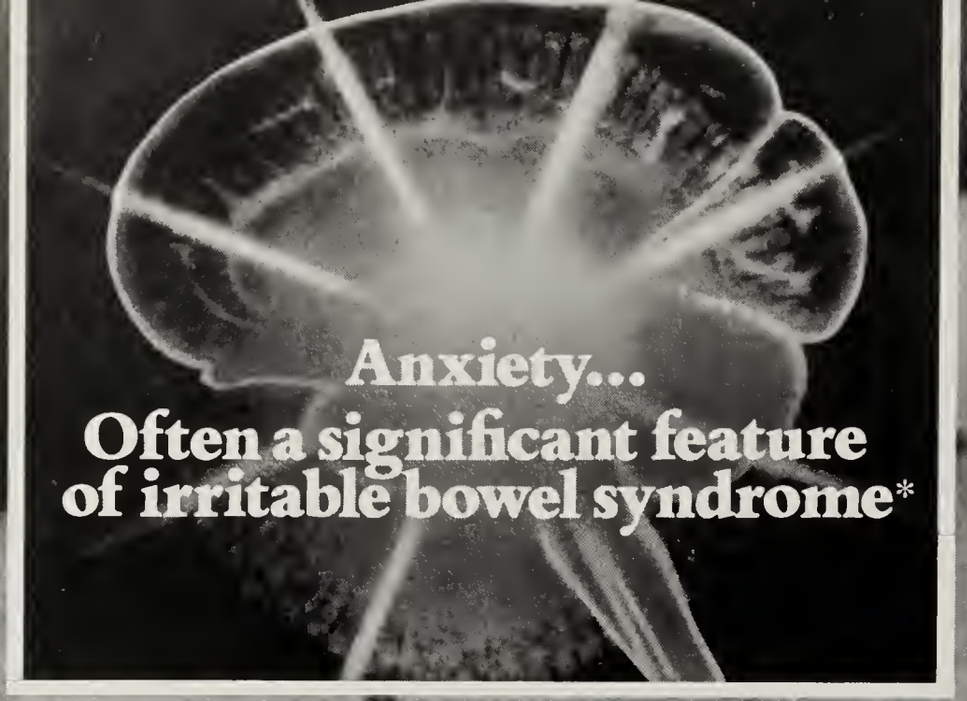
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*Librax has been established as possibly effective for this indication.
Please see brief summary of prescribing information on following page.

ROCHE

Librax®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium Br.

Please consult complete prescribing information, a summary of which follows:

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered, isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

ROCHE

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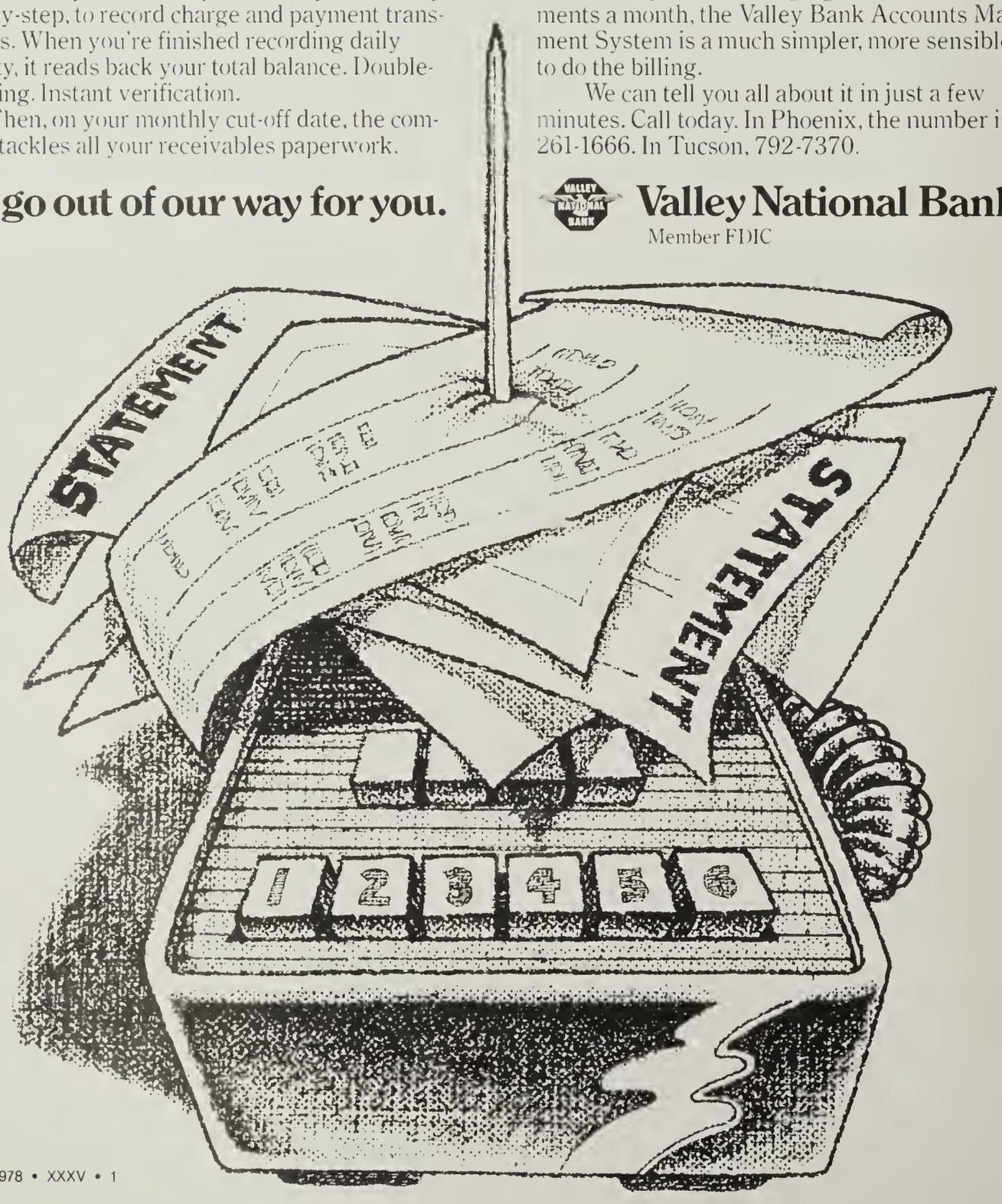
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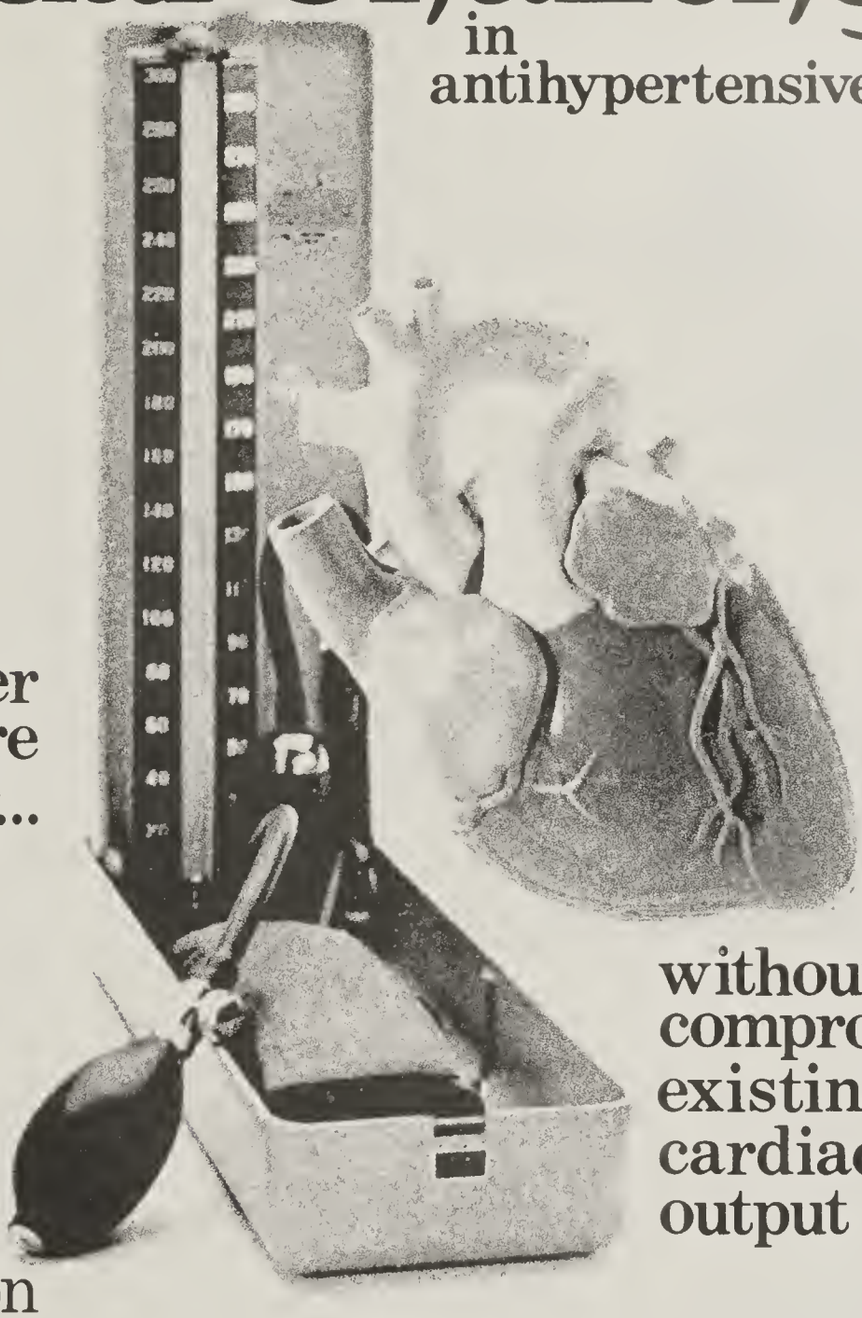
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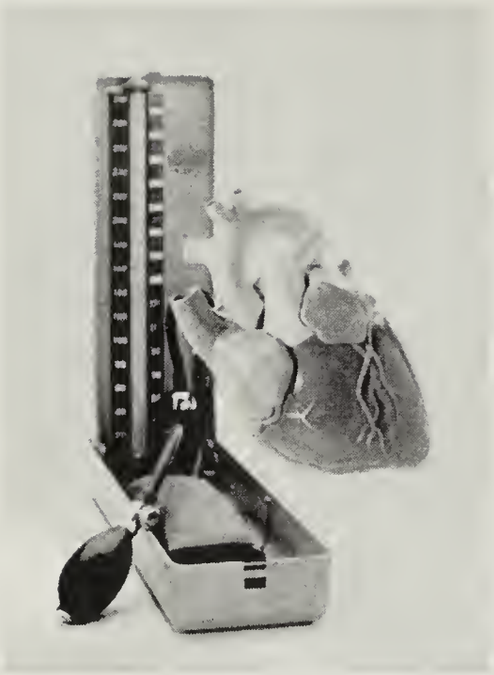
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ALDOMET is contraindicated in active hepatic disease, hypersensitivity to the drug, and if previous methyldopa therapy has been associated with liver disorders.

It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. For more details see the brief summary of prescribing information.

For a brief summary of prescribing information, please see following page.

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in hypertension

ALDOMET®

(METHYLDOPA|MSD)

helps lower blood pressure effectively... usually with no direct effect on cardiac function– cardiac output is usually maintained

Contraindications: Active hepatic disease, such as acute hepatitis and active cirrhosis; if previous methyldopa therapy has been associated with liver disorders (see Warnings); hypersensitivity.

Warnings: It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions.

With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood.

At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstituted. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, occasionally with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstituted in such patients.

Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

Pregnancy and Nursing: Use of any drug in women who are or may become pregnant or intend to nurse requires that anticipated benefits be weighed against possible risks; possibility of fetal injury or injury to a nursing infant cannot be excluded. Methyldopa crosses the placental barrier, appears in cord blood, and appears in breast milk.

Precautions: Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: urinary uric acid by the phosphotungstate method, serum creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetotic movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyldopa because the drug is removed by this procedure.

Adverse Reactions: *Central nervous system:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetotic movements; psychic disturbances, including nightmares and reversible mild psychoses or depression.

Cardiovascular: Bradycardia, aggravation of angina pectoris. Orthostatic hypotension (decrease daily dosage). Edema (and weight gain) usually relieved by use of a diuretic. (Discontinue methyldopa if edema progresses or signs of heart failure appear.)

Gastrointestinal: Nausea, vomiting, distention, constipation, flatus, diarrhea, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis.

Hepatic: Abnormal liver function tests, jaundice, liver disorders.

Hematologic: Positive Coombs test, hemolytic anemia. Leukopenia, granulocytopenia, thrombocytopenia. Positive tests for antinuclear antibody, LE cells, and rheumatoid factor.

Allergic: Drug-related fever, lupus-like syndrome, myocarditis.

Other: Nasal stuffiness, rise in BUN, breast enlargement, gynecomastia, lactation, impotence, decreased libido, dermatologic reactions including eczema and lichenoid eruptions, mild arthralgia, myalgia.

Note: Initial adult dosage should be limited to 500 mg daily when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third months of therapy; increased dosage or adding a diuretic frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

How Supplied: Tablets, containing 125 mg methyldopa each, in bottles of 100; Tablets, containing 250 mg methyldopa each, in single-unit packages of 100 and bottles of 100 and 1000; Tablets, containing 500 mg methyldopa each, in single-unit packages of 100 and bottles of 100 and 500.

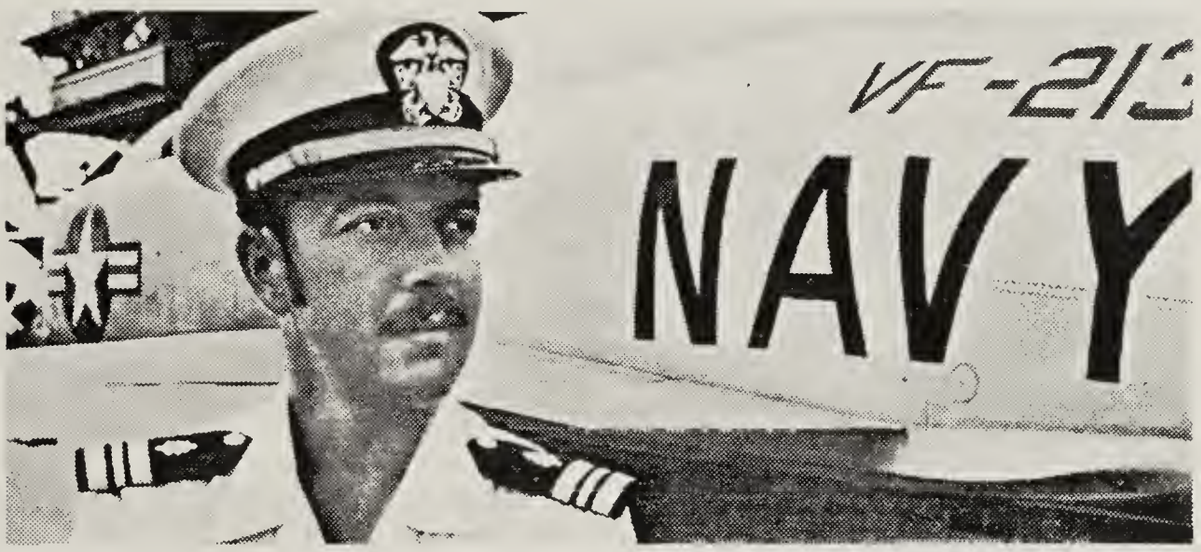
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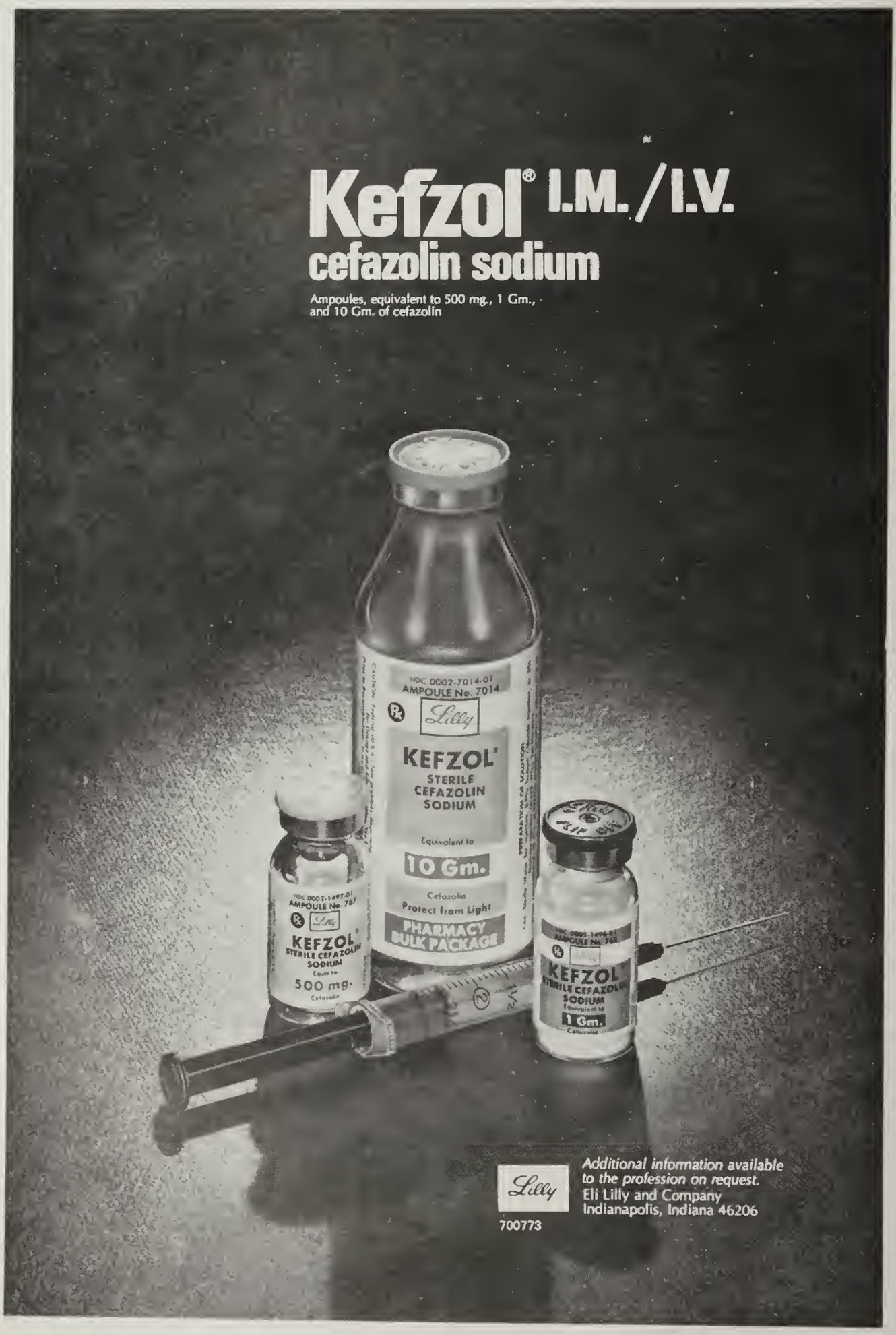
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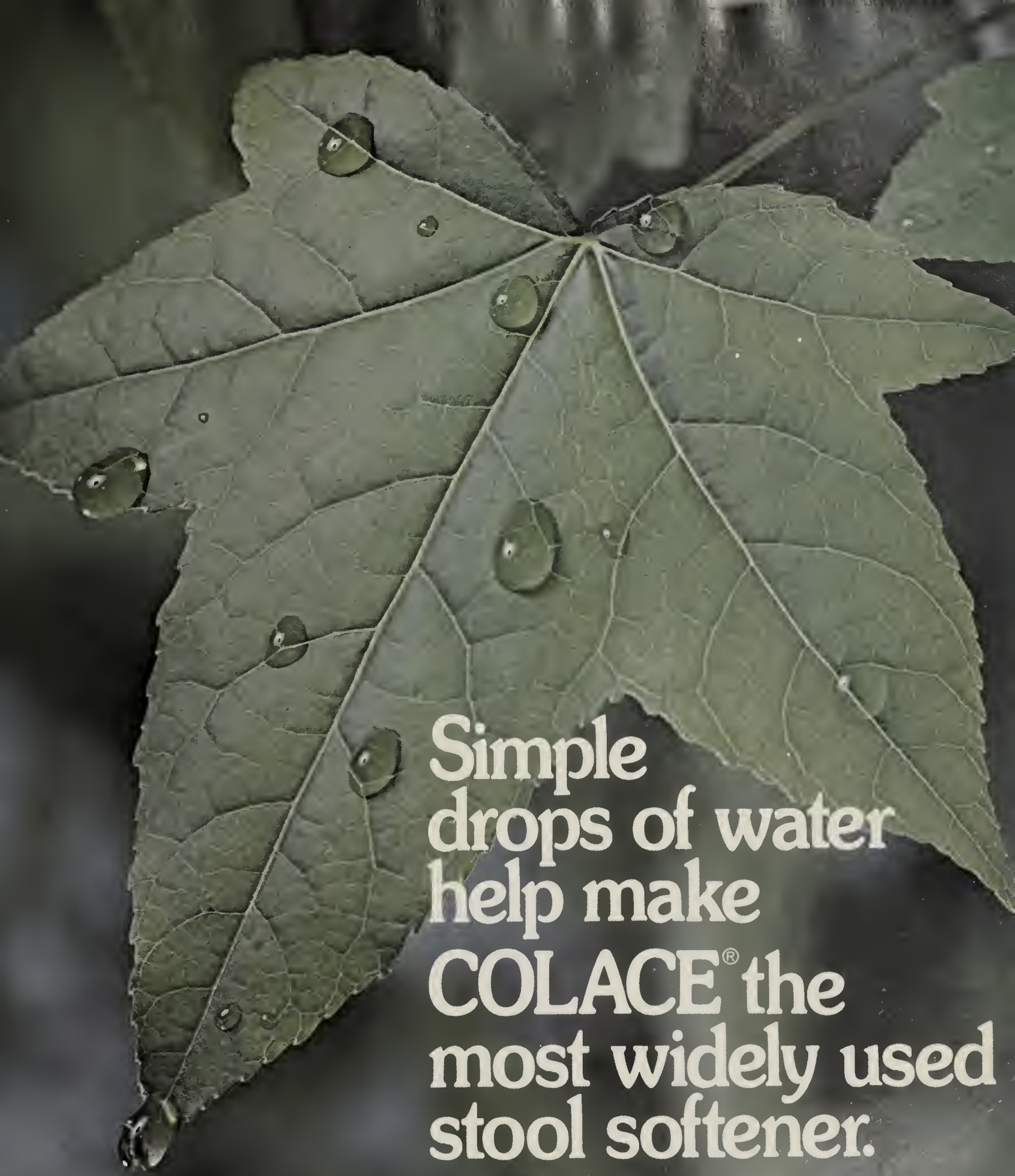
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Vasodilan–compatible with your total regimen for vascular insufficiency

Vasodilan can be a valuable adjunct in planning a total therapeutic program for vascular insufficiency.

*Indications: Based on a review of this drug by the National Academy of Sciences-National Research Council and/or other information, the FDA has classified the indications as follows:

Possibly Effective:

1. For the relief of symptoms associated with cerebral vascular insufficiency.
2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.

Final classification of the less-than-effective indications requires further investigation.

Composition: Vasodilan tablets, isoxsuprine HCl, 10 mg. and 20 mg. Vasodilan injection, isoxsuprine HCl, 5 mg., per ml.

Dosage and Administration: Oral: 10 to 20 mg., three or four times daily. Intramuscular: 5 to 10 mg. (1 or 2 ml.) two or three times daily. Intramuscular administration may be used initially in severe or acute conditions.

Contraindications and Cautions: There are no known contraindications to oral use when administered in recommended doses. Should not be given immediately postpartum or in the presence of arterial bleeding.

Parenteral administration is not recommended in the presence of hypotension or tachycardia.

Intravenous administration should not be given because of increased likelihood of side effects.

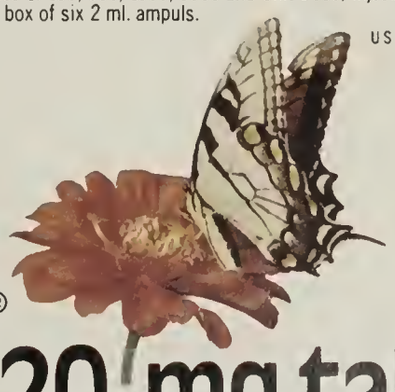
Adverse Reactions: On rare occasions oral administration of the drug has been associated in time with the occurrence of hypotension, tachycardia, nausea, vomiting, dizziness, abdominal distress, and severe rash. If rash appears the drug should be discontinued.

Although available evidence suggests a temporal association of these reactions with isoxsuprine, a causal relationship can be neither confirmed nor refuted.

Administration of single dose of 10 mg. intramuscularly may result in hypotension and tachycardia. These symptoms are more pronounced in higher doses. For these reasons single intramuscular doses exceeding 10 mg. are not recommended. Repeated administration of 5 to 10 mg. intramuscularly at suitable intervals may be employed.

Supplied: Tablets, 10 mg., bottles of 100, 1000, 5000 and Unit Dose; Tablets, 20 mg., bottles of 100, 500, 1000, 5000 and Unit Dose; Injection, 10 mg. per 2 ml. ampul, box of six 2 ml. ampuls.

U S Pat No 3,056,836



VASODILAN® 20-mg tablets

(ISOXSUPRINE HCl)

20 mg q.i.d. recommended dosage

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Indications: For the symptomatic treatment of bronchospastic conditions such as branchial asthma, asthmatic bronchitis, chronic bronchitis, and pulmonary emphysema.

Dosage: Initial: Adults: 1-2 capsules or 1-2 tablespoonfuls elixir every 6-8 hours, children 8-12: 1 tablespoonful or one capsule every 6-8 hours and children under 8: 3 to 5 mg theophylline/kg body weight every 6-8 hours. Theophylline dosage may be cautiously increased to 2000 mg/24 hr in adults or 7 mg/kg in children; monitoring of serum theophylline levels at higher dosages is recommended.

Precautions: Do not administer more frequently than every 6 hours, or within 12 hours after rectal dose of any preparation containing theophylline or aminophylline. Do not give other xanthine derivatives concurrently. Use in case of pregnancy only when clearly needed.

Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea and vomiting. The frequency of adverse reactions is related to the serum theophylline level and are not usually a problem at serum theophylline levels below 20μg/ml.

How Supplied: Capsules in bottles of 100 and 1000 and unit-dose packs of 100; Elixir in bottles of 1 pint and 1 gallon.

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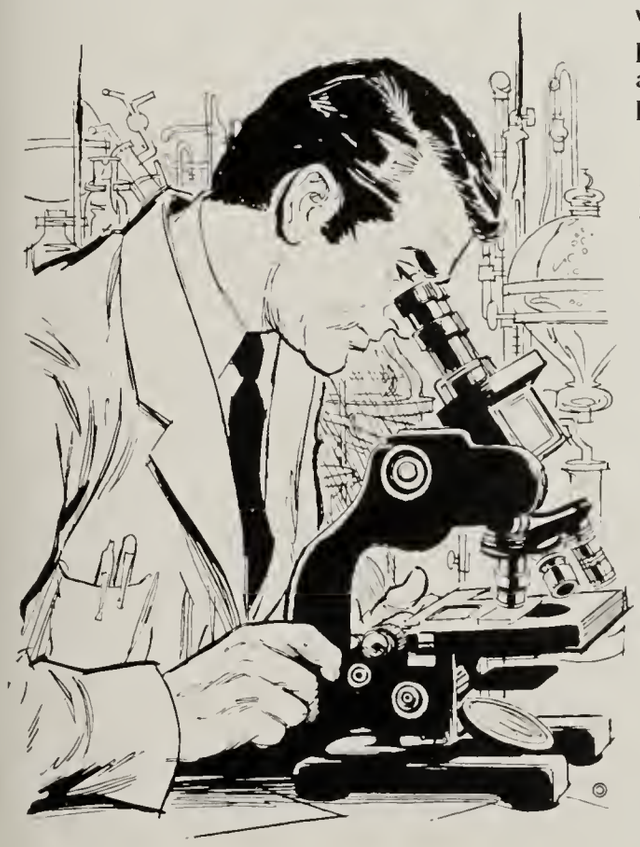
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Drug-Induced Systemic Lupus Erythematosus

William N. Jones, B.S.
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Philip D. Walson, M.D.

Systemic lupus erythematosus is a collagen vascular disease of unknown etiology whose cardinal manifestations are secondary to small vessel vasculitis. Although considered an autoimmune inflammatory disease, this classification is not revealing in terms of etiology.

Both idiopathic and drug-induced forms of the disease are recognized. Arthritis and arthralgia are the most frequent presenting symptoms along with fever, nephritis, rashes and seizures.[23, 41] Clinical manifestations occurring during the course of the disease are shown in Table 1. Although the features of drug-induced and idiopathic SLE are for the most part identical,[37] it is difficult to clinically separate individual cases. However, minor differences in the incidence of signs and symptoms have been observed. For example, with idiopathic SLE, arthritis and arthralgia occurred as presenting symptoms in 53% of the patients and in 92% during the course of the disease.[18] In contrast, arthritis and arthralgia occurred as the primary presenting symptoms in 60% of hydralazine-induced and 65% of procainamide-induced SLE. During the course of the disease the incidence of arthritis and arthralgia was also lower being 74% and 67% respectively.

Idiopathic SLE is primarily a disease of women, usually in the childbearing age group. In the drug-induced form of SLE the female is still affected more often than the male, but the incidence is about 3 to 2 instead of 9 to 1.[37] The age of the patient who develops drug-induced SLE is related to the disease being treated (i.e., anticonvulsants are responsible fo most pediatric cases while hydralazine and procaina mide induced SLE is more prevalent in adults)[2] an there tends to be a lesser incidence of renal and CN involvement with the drug-induced form.[1, 25, 37]

TABLE 1
Major Manifestations of SLE

Manifestation	Incidence (%)	
	Estes and Christian[23]	Dubois[18]
Arthritis and Arthralgia	95	91.6
Fever	77	83.5
Cutaneous reactions	81	71.5
Neuropsychiatric	59	25.5
Renal involvement	53	46.1
Pulmonary involvement	48	30.3
Cardiac involvement	38	30.5

The diagnosis of drug-induced SLE is based on th American Rheumatic Association's criteria for idio pathic SLE[13, 51] with two additional generalizations (1) the drug must be administered prior to the onset o any symptoms of SLE; and (2) the syndrome must b reversed upon discontinuation of the offendin agent[26, 37]

Serologic testing is often useful in diagnosing SLE. Significant differences have been deomonstrated between the drug-induced and idiopathic syndromes. Idiopathic SLE[3] and hydralazine-induced SLE[47] are generally associated with antibodies to native DNA (anti-double strand DNA antibodies). Antibodies to native DNA are generally not induced by procainamide,[2] INH,[3] anticonvulsants,[3] or the phenothiazines.[19] Even when clinical symptoms improve within

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TABLE 2
Drugs Associated with Activation of SLE-Like Syndrome

llopurinol	Oral Contraceptives
lpha-methyldopa	Oxyphenbutazone
minosalicylic Acid	Penicillin
arbamazepine	Phenylbutazone
hlorpromazine	Phenytoin
lofibrate	Practolol
orticosteroids	Prazosin
orticosteroid Withdrawal	Primidone
)-Penicillamine	Procainamide
thosuximide	Propranolol
old Compounds	Propylthiouracil
uanoxan	Quinidine
lydralazine	Reserpine
soniazid	Streptomycin
evodopa	Sulfonamides
lephenytoin	Tetracycline
lesantoin	Trimethadione
lethylthiouracil	Vitamins
lethysergide	

erologic findings may not change for onths [28, 29, 36, 37] or even years. [16] Another feature of oth the idiopathic and drug-induced syndromes is a alse-positive serologic test for syphilis [1, 28, 31, 43] hich will return to a negative serologic test in the rug-induced form when the drug is withdrawn. [43]

Although over 300 cases of drug-induced SLE have een reported, representing at least 36 drugs (Table), many reports have been poorly documented. Even o, there have been numerous attempts to categorize rugs suspected of inducing lupus syn- rome. [1, 25, 28, 37] Regardless of how the syndrome is escribed, the most consistent feature is chronic use of hese drugs. [25]

The mechanism by which drugs initiate, worsen, or ctivate SLE is not known. [1, 17, 22, 25, 28] A variety of nechanisms have been proposed as being responsible or the development of rash, pleuritis, alopecia and ther symptoms. [2, 22, 25] A predisposition to the levelopment of a lupus syndrome seems appar- nt. [1, 3, 4, 10, 53] Recently, however, the belief in a upus diathesis has been proposed to include a redisposition to autoimmune disease of various ypes. Alpha-methyldopa supports this theory since it as been implicated in causing SLE, but much more ften causes an autoimmune hemolytic anemia. [2]

Hydralazine and procainamide have been associated with more than two-thirds of the over 300 cases of drug-induced SLE reported. In fact, these drugs have been used as experimental models of the syndrome itself. Since the best documented and largest number of cases of drug-induced SLE have been associated with these two drugs they will be discussed in detail. Drugs associated with inducing SLE, but not as well documented, will be briefly presented.

Hydralazine

The first cases of a hydralazine-induced disease simulating a collagen disease were reported in 1953 and confirmed in 1954. [21, 44, 48] The incidence of this so-called "hydralazine syndrome" has been reported to be between 2 and 21% of those individuals treated with hydralazine. [9, 37, 48] The incidence appears to be dependent on dose and duration of treatment. A dose of 400 mg or more per day is more often associated with "hydralazine syndrome," expecially when taken for periods of three months or longer. However, of 50 patients who had febrile, cutaneous or rheumatic reactions while taking hydralazine, 19 were taking 200 mg or less daily and one was taking 75 mg per day. [4] The syndrome occurs on an average of 17 months from the onset of therapy, but it has been noted in patients taking hydralazine for as short as three weeks [37] or as long as 10 to 20 years. [6, 47] The variance in dose and the incidence of drug-induced lupus has led to the hypothesis of a predisposition to SLE in patients who develop the syndrome [1, 4, 5, 37] In 74% of 50 patients with "hydralazine syndrome" a history suggestive of a predisposition was found, while in only 13% of 100 patients with hypertension the evidence of a lupus diathesis existed. [1]

Perry et al. [47] reported an inverse relationship between the symptoms of drug-induced SLE and the blood pressure response to hydralazine demonstrating that, as symptoms worsened, a significant improvement in hypertension was observed. They also observed that patients who developed an early febrile reaction when treated with hydralazine developed SLE more often.

Several signs and symptoms are associated with hydralazine-induced SLE. A false-positive serologic test for syphilis, recurrent, migratory thrombophlebitis or Raynaud's phenomenon may be the only manifestations of the diathesis. [4] Renal involvement has been observed in 20% of cases and LE cells were more prevalent in hydralazine-induced SLE than in the idiopathic disease. [1] Pericarditis occurs in about 2% of the cases of idiopathic SLE [1] and pericardial tamponade has been reported with hydralazine-induced SLE. [12]

Procainamide

Procainamide is the most common cause of drug-induced SLE. Ladd reported the first case of procainamide-induced SLE in 1962 [36] and numerous authors have confirmed the report. [14, 15, 20, 46]

The dosage required for procainamide-induced SLE varies from a total of 14 to 3600 g [37] with a daily dose of as little as 500 mg having been reported to cause the syndrome. [7] The time from initial therapy to the onset of symptoms is usually three months to two years of continuous administration with the average being slightly more than six months. Individual cases, however, have been recorded where the initial symptoms appeared as early as two weeks to as late as

four and one-half years from initiation of therapy.[37]

Pleuritis, unique to procainamide-induced SLE, has been reported in about 30% of cases.[1] Pleuritic or pericardial chest pain was the second most consistent symptom and was reported in 8 of 12 patients who developed procainamide-induced SLE. Of these 8 patients 4 developed pulmonary infiltrates and 2 developed pleural effusions.[46] Pulmonary involvement in drug-induced SLE is limited to procainamide, and it is believed that the patient population may be related to this manifestation.[11]

Evidence exists for a diathesis in procainamide-induced SLE.[34] Reasons include the wide range of doses found to induce SLE**37** and the occurrence in siblings.[45] Also suggesting some predisposition is a positive ANA which commonly appears in patients taking procainamide, while only about 5% of patients taking the drug for more than 6 months will actually develop the syndrome.[9, 25, 35, 37]

Other Antihypertensive Drugs

Reserpine has been implicated in inducing SLE. In a retrospective study Lee et al.[38] found only 2 cases of reserpine-induced SLE out of nearly 1200 patients. This infers that reserpine is a weak inducer of SLE if at all. A review of the literature up to 1975 does not yield any additional reports of SLE induced by reserpine.[37]

Alpha-methyldopa is known to cause a direct positive Coombs test in about 20% of patients treated; of those, only 2% develop hemolytic anemia.[40] In a report of three cases of Coombs positive hemolytic anemia two patients developed LE cells and rheumatoid factor without further evidence of SLE or rheumatoid arthritis.[52] Other authors have demonstrated LE cells and ANA in patients without evidence of SLE[30, 40] leading Lee and Chase[37] to conclude that no patients have developed a lupus syndrome secondary to methylodopa. On the other hand, Alarcon-Segovia implicated methyldopa as causing an increase in all autoimmune disorders and not just SLE.[2]

Other Cardiac Drugs

Although quinidine has been implicated as an activator of SLE, conclusive evidence of quinidine's inclusion as an inducer of SLE is lacking.[19, 37]

Practolol[37, 49] and propranolol[29] have been claimed to induce SLE, but the cases involving practolol do not meet the definition of Lee and Chase[37] of drug-induced lupus since corticosteroids were required to reverse symptoms. Propranolol has been implicated in one patient who was also taking hydralazine. Hydralazine was withdrawn when the patient developed SLE manifestations, but the symptoms did not remit until the propranolol was withdrawn.[29]

Antituberculosis Drugs

Nine years after the introduction of isoniazid in 1952 the first cases of an SLE syndrome were reported.[37] It is difficult to assess the status of INH-induced SLE since it is common procedure to treat tuberculosis wit several drugs, including PAS and streptomycin, whic have also been implicated in inducing SLE.[1, 2, 19, 37]

Symptoms have appeared in patients taking INH a soon as four weeks after continuous therapy and up t as long as 14 months from initial treatment. Dosag has ranged from a total of 25 to more than 100 g INH.[37] The induction of ANA is quite common bu the incidence of INH-induced SLE is rare.[19]

Anticonvulsants

All of the classes of anticonvulsants have bee implicated as inducers of SLE.[19, 37, 39] Furthermor the drug-induced lupus syndrome seen in children i most often associated with anticonvulsants (agai showing that the population affected by drug-induce SLE depends on the etiology of the disease bein treated).[8] Although about 60 cases of anticonvulsant induced SLE have been recorded,[1] the caus relationship has been questioned.[19] Since seizures ar an initial manifestation in about 3% of patients wit idiopathic SLE,[23] the implication of anticonvulsant inducing SLE is difficult to make.[37]

Antipsychotic Drugs

In a five-year study in two mental hospital antipsychotic-induced SLE was diagnosed eight time in a population of 4300 patients.[24] Other cases hav been documented, but the incidence is low. Patient taking chlorpromazine continuously for prolonge periods have antinuclear factors in their blood in abou 24% of cases.[19, 37] If symptoms of SLE appear the are usually mild (arthralgia) and improve gradually.[24]

Treatment

Treatment of drug-induced lupus is withdrawal o the offending agent from the therapeutic regimen Generally, symptoms will remit when the drug i withdrawn.[19, 37] If symptoms persist the diagnosis o idiopathic lupus must be considered.[37]

In very ill patients corticosteroids may be of benefi in accelerating recovery.[9, 37] However, there hav been two reported cases of corticosteroid-induce SLE. Symptoms of arthralgia, myalgia and pro teinuria increased when these patients were treate with corticosteroids. Only when the corticosteroid were withdrawn did the two patients improve.[27]

The question of whether to allow a patient who ha developed drug-induced lupus to remain on tha treatment has been argued.[32, 50] Perhaps it would b best to avoid the drug if possible[2] and weigh the risk versus gain in each case.

Summary

Drug-induced SLE is difficult to distinguish from idopathic SLE. Generally, a drug-induced lupus is a milder form of the disease. With few exceptions the disease usually worsens and symptoms become more severe the longer the medication is continued. By definition, the drug-induced form generally remits

hen the drug is withdrawn.[9, 37]

The manifestations of drug-induced SLE are ssentially identical to spontaneous lupus, except that nal and CNS involvement are usually absent. lowever, in hydralazine-induced SLE a 20% incidence f renal involvement has been reported.[2]

Any patient taking a drug implicated in inducing LE who develops any symptoms of SLE should have rug-induced lupus considered, especially if the anifestations include pericarditis, pleuritis or arthri-is. If possible, the suspected drug should be iscontinued at once. It would also prove helpful to ule out SLE serologically prior to starting therapy ith either hydralazine or procainamide to avoid iagnostic confusion if signs or symptoms of SLE ppear.

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Emotional Disorders

Clinical Depression Complicating Chronic Disease

Stephen B. Shanfield, M.D.

Many, but not all, chronically ill patients are emotionally depressed, since they have a depressed mood or affect, plus two of the following: sleep disorder, excessive weakness, increased dependency feelings, helplessness and hopelessness; or if there is significant suicide content. Psychiatric consultation is advisable if the patient shows delusions or a high suicide content. Treatment of this depression consists of energetic medical management, including antidepressant medication, setting realistic rehabilitative goals with encouragement of successes and structured group activities.

Emotional depression is frequently associated with chronic medical and surgical disease,[3] when "chronic" is defined as being irreversible and of long duration.[1,2] The emotional depression may account for most of such patients' impairment of function. What are the signs and symptoms of the depression which appears in patients with chronic disease, and what treatment is effective?

For a depression to be of clinical significance, there must be both a subjective depressed affect—which is a mood or feeling—that is "sad, blue, down, unhappy" which appears in normal people, and also 2 or more objective phenomena, lasting longer than two weeks, from this group: Sleep difficulty or sleeping too much, appetite disorder with weight loss or gain, energy disorder with complaints of weakness, psychomotor agitation or retardation, loss of interest in usual activities, loss of sexual interest or function, feelings of excessive or inappropriate guilt, excessive crying, giving a negative coloration to the past, present or future, subjective or objective slowing of thinking processes or of logical thinking, excessive dependency feelings and suicidal thoughts.[3,4,5]

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Such a clinical depression is not another expected aspect of chronic illness, because some patients adapt to chronic illness without any discernible depression. Often the depression results from perceived major losses in the patient's social, psychological, economic, or physical functioning. The depressed chronic patient may feel weaker than is expected, may spend excessive time in bed and become socially withdrawn. Some chronic patients with pain may transmute their psychological suffering into an increase in the severity of pain, or into a new pain, while in others, the pain may provide a diversion from life's problems including the medical and surgical difficulties which they face.[6,7,8] If the depression is permitted to become a way of life for the chronic patients, it can be fused into the chronic disease pattern.[10] Hopelessness and helplessness in such patients may be imparted to other persons in their environment.[9]

TREATMENT

Fundamental to the treatment of the depressed chronic patient is the giving of a continuity of car support and hope. The physician must treat, admini ter and urge his patient to help replace the li sentence of despair with a life of hope. The physicia must also preserve his objectivity and sense of humo since many depressed patients have a need to t frustrating and to provoke anger and hostility.

Analgesic medication, when given p.r.n., tends t reinforce pain.[11,12] Antidepressant medication, suc as amytripiline or imipramine, however, is extremel valuable, using small dosages for older and brair injured patients.

Group activities are very helpful, expecially in olde patients. The successful accomplishment of tasks, a first small ones, then increasingly larger, can refor an individual's sense of mastery, thus helping him t build self esteem.

Psychiatric consultation is necessary in chronicall ill patients if there are delusions, insufficient respons to treatment, or if there is a significant suicid content, or a family history of suicide.

Seminars in Endocrinology and Metabolism

Paget's Disease: An Update on Therapy

Stephen E. Dippe, M.D., F.A.C.P.

Paget's disease was described by Sir James Paget in 1876. He initially gave it the term osteitis deformans. In spite of the long history of the disease, we still have no explanation as to its etiology. Paget's seems to occur more frequently in various sectors of our world, i.e., Central Europe, Australia, New Zealand and the United Kingdom. It is less frequent in Scandinavians and Americans and is rare in most of the other parts of the world. The frequency of the disease also increases dramatically with increasing age, (10% of those in the ninth decade have the disease[1]). There may be 2,500,000 cases of Paget's disease in the U.S.A. but only 5% have serious enough disease to require specific treatment.[2] The genetic characteristics of this disease remain obscure, and the familial occurence was less than 2% of the cases in a recent survey.[3]

Most patients with Paget's disease are asymptomatic. They seek medical attention for disfiguration of the lower extremities, pain, increasing head size, reduction in hearing, reduced mobility, stooped stature, warmness over the affected bones, fracture, and neurological defects. Associated with these symptoms are the clinical signs of bony involvement of the pelvis, spine, skull and femur. The disease is

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haracterized by osteoclastic reabsortion of normal one. The new bone that is made is irregular, lisorganized, and weak. There can be encroachment of he spinal cord causing paraplegia, encroachment of he 8th nerve causing deafness, and weakening of the one structure itself causing fractures. Since the bone s highly vascular, the patients can experience high utput cardiac failure. Also, there is an increased ncidence of sarcomatous degeneration within this bnormal bone.

A clear understanding of the etiology of Paget's lisease is absent. In the past, therapy has been poor nd has usually required the use of anti-inflammatory gents to combat pain. Clinical manifestations of the lisease have been used to follow the success of therapy n the past. Now that we have other modes of therapy hat definitely affect the disease process itself, we can se biochemical tests to determine the success or onsuccess of treatment. A specific isozyme of Alkaline phosphatase is liberated from bone and is xtremely high in many patients with Paget's disease. Also, the collagen metabolite, hydroxyproline, is xcreted in higher concentrations in the urine in this ondition. In order to substantiate the diagnosis of Paget's disease these supporting studies should be btained. Obviously, clinical signs, especially early igns, are not clearly diagnostic of Paget's disease. The use of x-rays is important, as very characteristic esions are seen in the skull and hips.

Calcitonin has been shown in numerous studies[4,5,6] o be quite effective in treating Paget's disease. Calcitonin is a hormone liberated by the parafollicular ells of the thyroid gland that depresses or inhibits steoclastic action and thus, slows the progress of the lisease. The success, however, is not only in the eduction of symptoms, but also in objective clinical nd laboratory evidence of improvement of the lisease. Bone biopsies have shown normal new bone ormation. When calcitonin is given, decreases in erum alkaline phosphatase occurs within two or three veeks and urinary levels of hydroxyproline levels fall. Within two or three months the levels may fall to the normal ranges. This, of course, is in the most favorable response. Radiologically, there is improvement.[7] Most important to the patient, however, is hat pain is relieved. As far as it is known, calcitonin has no effect on normal bone. The calcitonin that is available commercially is synthetic salmon calcitonin (Calcimar-Armour).

A major stumbling block in the use of calcitonin is the length of time the drug remains effective. The use of this drug has been present for a number of years and studies demonstrate that its effectiveness can last up to one, two and perhaps three years. Unfortunately, this is not present in every individual. Investigators have shown that neutralizing antibodies coincide with the relapse of the disease, both clinically and chemically. The side effects of the drug are really quite benign in that they consist of nausea, flushing and rashes.

Indications for calcitonin therapy depend on the individual and his disease process. Since most cases are asymptomatic and may remain asymptomatic for years without any complicating factors, it's obvious that these patients should not be given calcitonin. Treatment indications depend on the patient himself. If cardiac status is good, mobilization is excellent, pain is minimal, hearing is quite adequate, treatment should be withheld in these individuals until significant symptoms occur. Incidental findings on x-rays demonstrating Paget's disease should be followed periodically and the patient should be observed for any of the clinical findings associated with Paget's disease. If pain should become a major factor affecting his daily living, then calcitonin should be considered in the treatment of his disease. Of course, anytime a patient with Paget's disease is immobilized for fractures, careful observation of the calcium should be maintained and if hypercalcemia develops, then treatment with calcitonin would be beneficial. Treatment will help to prevent further deterioration of hearing and neurological defects.

Other forms of treatment include the use of diphosphonates[8] and mithramycin.[9] These drugs have been used successfully in the treatment of Paget's disease, but they have significant side effects.

One word of caution is necessary in evaluating patients with Paget's disease. Other arthritis conditions can occur concomitantly with Paget's disease.[10] Since pain may be a similar complaint in all of these conditions it is essential to determine the cause of the pain prior to instituting therapy.

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Introduction

Harold C. Tretbar, M.D., FACP

Editor Arthritis Issue

Arizona has always been a mecca for patients with arthritis. Blessed by its climate, Arizona has attracted both the patient and the well trained physician to care for him. Recently we have gained National recognition for our outstanding treatment and teaching programs.

Tucson has just had the honor of being named a Multipurpose Arthritis Center by the National Institutes of Health. The Federal Arthritis Act of 1975 directed the establishment and funding of arthritis centers. Out of 86 applications, Tucson was fortunate enough to be one of the 14 centers chosen.

In April, 1977 the American College of Physicians in conjunction with the University of Arizona School of Medicine sponsored a very successful Post-graduate Course entitled, "Clinical Rheumatology: The Diagnosis and Treatment of Arthritis and Related Diseases".

Phoenix hosted the Western Regional Section of the American Rheumatism Association in November. There was a combined program of review sessions and basic research reports.

Highlights of these meetings included the role of crystals in degenerative joint disease, the relationship of tissue typing antigens (HLA) to spondyloarthropathies and the use of Penicillamine to treat rheumatoid arthritis. Immuno-modulation was an area of intense interest. It seems peculiar that stimulation of the lymphocytes by agents such as levamisol or transfer factor may be just as effective in reducing inflammation as is suppression of the lymphocyte with cytotoxic drugs.

This issue of *Arizona Medicine* contains several articles that reflect our experience in treating a variety of joint diseases. These articles have been contributed by some of the faculty of the American College of Physicians Postgraduate Course. They were chosen to offer a balance between concepts of etiology, newer therapeutic approaches and treatment of often confusing every day problems.

Osteoarthritis

H. Ralph Schumacher M.D., FAC

INTRODUCTION

Osteoarthritis or degenerative joint disease is the most common type of arthritis. It is typically manifested clinically by pain on use of a joint, loss of motion, effusion and crepitus. Histopathologic findings include diminution in amounts of glycosaminoglycans, less chondrocytes, fraying and fissuring of cartilage and osteophyte formation. It seems certain that many different factors are involved in the pathogenesis of this clinical and pathologic picture. Some osteoarthritis appears to be an end result of structural injury to the joint. Metabolic changes in collagen or glycosaminoglycans, enzyme release from chondrocytes and other cells, changes in subchondral bone, and alterations in lubrication may also be involved. Familial factors are important in some instances, as in the primary osteoarthritis characterized by Herberden's nodes.

Inflammation clearly can be a component of osteoarthritis. Peter et al[1] have shown histologic evidence of acute inflammation in Herberden's nodes. Synovial effusions in osteoarthritis although usually considered as "non-inflammatory," have leucocyte counts up to 3,600/mm^3 in the series of Ropes and Bauer.[2] Mechanisms for this inflammatory aspect of osteoarthritis have not been defined. Since crystals of calcium pyrophosphate dihydrate[3] and hydroxyapatite[4,6] have now been identified in osteoarthritic effusions, they merit strong consideration as causes of some of the signs of inflammation and clinical symptoms of osteoarthritis. That cartilage crystal deposits are factors in early cartilage damage is also a possibility. Search for crystals in osteoarthritic joints is an active area of current research.

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he Role of Articular Crystals

alcium Pyrophosphate Dihydrate Crystals [CPPD]

Approximately 50% of patients with chondro-lcinosis in whom CPPD crystals are found have atures of osteoarthritis.[3] It is not yet established if is degenerative joint disease is a result or a cause of e crystal deposition. Studies from Czechoslovakia ow x-ray evidence of chondrocalcinosis to precede y radiographically identifiable degenerative anges.[7] Our electron microscopic studies of chondro-lcinosis patients from the Chiloe Islands of Chile[8] lentified subtle, probably degenerative, changes in ondrocytes from articular cartilages that had no vidence of crystal deposition.

There is as yet no prospective study in the USA to y to determine if the crystals cause and/or are used by degenerative changes in cartilage. The istribution of joint involvement in full-blown chon-rocalcinosis and osteoarthritis differs from that in rimary osteoarthritis[3] suggesting participation of ther factors. Patients with CPPD deposition disease ave prominent involvement of wrists, MCP joints, bows and ankles in addition to knees and hips. DIP oints and 1st MC-C joints as in primary osteoarthritis an be involved with osteoarthritis in patients with PPD but the frequency of CPPD in such patients has ot been established. Chondrocalcinosis appears to ncrease with age (as does osteoarthritis). Chondrocal-inosis was found in 27.6% of residents of a Jewish ome for the aged.[9] Howell et al[10] have shown that steoarthritic cartilage releases more pyrophosphate han normal in tissue culture. Osteoarthritic effusions nd synovial fluids from other diseased joints have levated levels of pyrophosphate compared to ormal.[11]

The apparent association with CPPD of a number of metabolic diseases (hemochromatosis, hypothyroidism, hyperparathyroidism, ochronosis) capable of altering connective tissue metabolism raises the possibility that a matrix or chondrocyte (degenerative?) change might antedate crystal deposition in the idiopathic cases. The granular glycosaminoglycan-rich matrix surrounding CPPD deposits[8] may be a late change or an early matrix alteration that favors crystal deposition. Some of the smallest CPPD crystals are lined up on collagen fibers[4] suggesting that a change at the collagen fiber might be important. We have seen apatite but not CPPD associated with matrix vesicles.

CPPD crystals are characteristically 3-15 μm, weakly positively birefringent, rod or rhomboid shaped crystals. They are most often intracellular during acute inflammation and may also be seen in phagosomes of synovial fluid mononuclear cells in the absence of an increased leucocyte count in synovial fluid.[12] By electron microscopy, crystals are dark with a foamy internal structure.[4, 8, 20] Some very small examples of CPPD might be visible as crystals only by electron microscopy (EM).

CPPD crystals should be sought in all patients felt to have degenerative joint disease (DJD) with acute inflammatory exacerbations. The frequency of CPPD crystals causing such bouts is not known but that they do occur is clear. Don't forget that you need not see chondrocalcinosis by x-ray. Deposits may be too small to be visible radiographically or all cartilage may be destroyed in advanced DJD with dispersal of the crystals (Fig. 1).

Apatite Crystals

Apatite crystals, the normal mineral of bone, have recently been demonstrated in osteoarthritic synovial fluids by scanning[6] and transmission[5] electron



Figure 1
Chondrocalcinosis seen well in the articular cartilages of the lateral aspect of the knee. The medial joint space shows joint space narrowing and less well defined chondrocalcinosis.

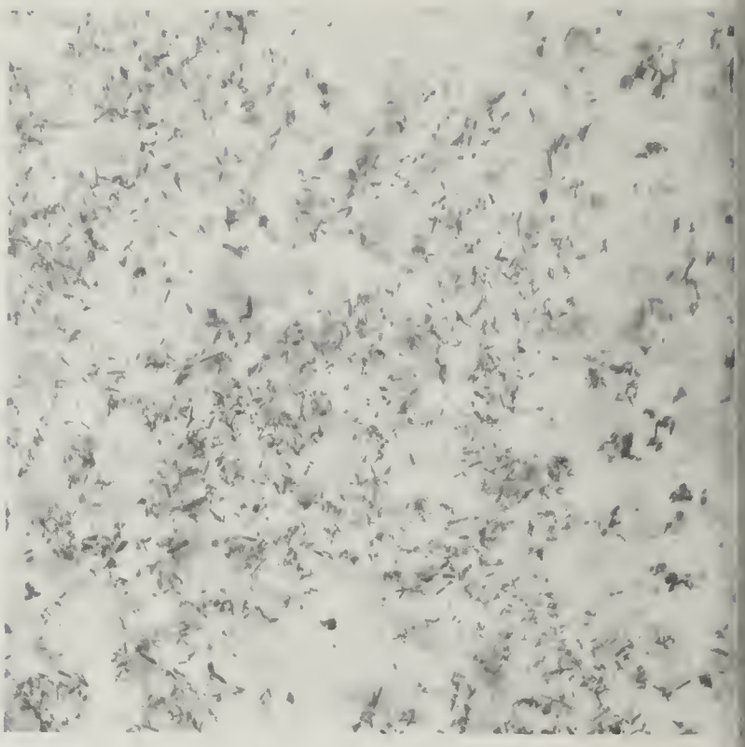


Figure 2
Electron micrograph demonstrating the tiny rod or needle shap apatite crystals visable in synovial fluid as crystals only by EM Note that most of these crystals lie in a yet unidentified granul matrix that may influence their properties in vivo. 20,000x.

microscopy with election probe analysis. The typical calcium: phosphorus ratio for apatite of about 1.6:1 has been found in all our samples. X-Ray diffraction has shown typical patterns of apatite in joint fluid in acute arthritis without osteoarthritis but we have not yet found enough crystals to do this in degenerative joint disease. We have now identified apatite crystals by EM and probe analysis in 6 of 12 osteoarthritic fluids studied. One patient with chondrocalcinosis and CPPD crystals also had apatite.

McCarty et al [13] also noted apatite deposits in occasional cadaver menisci but did not comment on whether there were any associated degenerative changes. Bennet, Waine and Bauer [14] in 1942 described granular hematoxyphilic deposits felt to be due to calcification in 3 menisci with degenerative changes. Howell has found hematoxyphilic chunks in osteoarthritic cartilage that may represent apatite. In experimental osteoarthritis, calcification has been observed in the fibrous cartilage-synovial junction and has been felt to be a precursor of osteophyte formation. [15] Schwartz of the University of Virginia has shown me calcification apparently due to apatite developing in tissue cultures of osteoarthritic cartilage.

Although in advanced osteoarthritis some apatite might be expected to come from bone, most crystals seen to date appear to be independent of bone fragments. Origin of the joint fluid crystals may well be the cartilage deposits. Mechanisms for crystal deposition in cartilage are not yet known nor is it known if degenerative changes allow the apatite t deposit and/or if the apatite contributes to th degeneration.

Crystals of apatite [4, 5] although rarely producin clumps that are birefringent with polarized light, ar generally not visible by light microscopy in ostec arthritic synovial fluids. Each crystal is only 75-250 A (Fig. 2) in diameter and is seen only by EM. By EM apatite needles in joint fluid are often lying in granular matrix and do not appear to be associate with collagen. Clumps of apatite may be seen by ligh microscopy in bursitis and acute arthritis as "shin coin" like bodies as large as RBC's but in osteoarthri tis these have been uncommon. Smaller shiny clump or hematoxyphilic and VonKossa positive globules ca suggest the presence of apatite in an osteoarthriti synovial fluid. Crystals are often extracellular but ca also be seen in synovial fluid cell vacuoles. Synovia fluid leucocyte counts in our osteoarthritic patient with apatite crystals have ranged form 100-900mm3 [5] The higher counts although not greater than th 2000/mm3 that is generally considered "inflammtory" may represent a mild cellular response to the crystals

Apatite can induce inflammation after injection int dog knee joints exactly as has been done with urate and CPPD. Not all effusions with any of these crystal are associated with inflammation [4]. Other factors such as coating of crystals with immunoglobulins, number and sizes of crystals, soluble materials etc. [4] are unde study to determine which of these are critical in inducing detectable inflammation.

As noted above, not all osteoarthritic effusions nor ven all with signs of inflammation demonstrate patire by our techniques [5] using transmission EM. Ve suspect that other factors account for some of the fusions but cannot be sure that improved techniques ill not show crystals in increasing numbers of fusions.

As with CPPD, calcification need not be visible by ray for apatite crystals to be present in a synovial fusion or in articular cartilage. One may see opaque pecks around osteoarthritic joints. The frequency and gnificance of these need to be determined.

ther Crystals

Calcium hydrogen phosphate dihydrate crystals ith strong positive birefringence have been known to cur in joints[13], [16] for several years. A recent report Gaucher et al [17] has identified these crystals as the parent cause of a destructive arthropathy in a tient with cartilage calcification on x-ray. It can be ticipated that further examples will be identified in e future.

reatment

Current understanding of any possible role of CPPD r apatite in pathogenesis of osteoarthritis does not llow any insights into propyhylaxis or therapy of the arly cartilage deterioration. If crystal deposition can e stopped or crystals removed as suggested with PPD by McCarty[2] one might be able to alter some of ne cartilage change. Supression of inflammation as iscussed below may help prevent some secondary rtilage changes if amounts of enzymes released from ucocytes can be decreased.

The crystals do appear to be one factor contributing n element of inflammation to osteoarthritis. In atients with acute "pseudogout" superimposed on steoarthritis, use of an anti-inflammatory drug such s indomethacin will clearly decrease the acute pain nd swelling.

Anti-inflammatory drugs have also been observed to ecrease pain in some patients with osteoarthritis who pparently were not felt to have obvious associated rystal deposition. Whether suppression of low-grade rystal induced inflammation is one explanation for nis remains to be seen.

In patients with known CPPD crystals accounting or their inflammation, indomethacin seems to be nsiderably more effective than aspirin. Some of the ewer nonsteroidal agents may also be useful. horough aspiration of the joint may be all that is equired in some cases.[3] Intra-articular steroid jections also may help. That supression of low grade rystal induced synovitis is one of the reasons that ntra-articular steroids help in osteoarthritis is an ntriguing possibility.

Patients with CPPD crystals occasionally appear to espond to colchicine but still early studies suggest hat apatite crystal associated inflammation may more frequently resolve with this agent.[5], [19]

Anti-inflammatory drugs of course may have additional effects beside the supression of the signs of inflammation. In in-vitro studies McKenzie et al [18] showed that several anti-inflammatory agents tended to decrease glycosaminoglycan synthesis in aged human cartilage.

Summary

Calcium pyrophosphate and apatite crystals can be found in articular tissues and fluid of many patients with osteoarthritis. These seem likely factors in the inflammation seen in many people with osteoarthritis. These and other crystals also need to be considered for a possible contribution to the early degenerative changes.

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Surgical Aspects of Arthritis of the Neck

Robert P. Goldfarb, M.D., F.A.C.S.

Neck discomfort including pain and stiffness is an extremely common complaint. Frequently these symptoms are secondary to simple muscular spasm, but they may be caused by arthritis of the cervical spine. The arthritis may be either osteoarthritis or rheumatoid arthritis. In diagnosing the etiology of neck discomfort one should not overlook the fact that tumors of the cervical region may present with symptomatology similar to arthritis.

It is not surprising that neck pain and its association with arm or shoulder discomfort ranks with headache as one of the most common sets of symptoms reported to the physician.

Patients who have neck pain from nerve root compression with radicular symptoms radiating to the arm and hand, must be distinguished from those patients who have long tract findings compatible with a cervical myelopathy.

It is useful to divide the pathological processes of the cervical spine into 1) Osteoarthritis (degenerative joint disease); 2) Rheumatoid disease; 3) Tumors of the cervical spine.

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OSTEOARTHRITIS

Osteoarthritis (degenerative joint disease) affect two distinct articular systems. The intervertebra articular and apophyseal articulations (interfacetal are the joint systems involved. The interfaceta articulations allow anterior and posterior glidin movement. Acute arthritis may involve one or more o the articulations with pain and limitation of motion The intervertebral articulations consist of an inter vertebral disc which is attached to the vertebra b hyaline cartilage. With aging or degeneration of th disc, the water content of the disc is reduced, as ar the protein-polysaccharide levels.[1] This results i decreased resiliency of the disc and the tendency t prolapse.

With degenerative narrowing of the disc space, th margins of the vertebral bodies approach each other Pressure and rubbing of these surfaces produce a eburnation of bone.

With these changes of osteoarthritis, neurologica syndromes may be seen. Marginal osteophytes ca narrow the spinal canal and compress the spinal cord The resultant myelopathy may cause gait changes sensory changes including numbness, spasticity increased reflexes, ankle clonus, and often positive to signs. On lateral cervical spine radiographs, the AP diameter (sagittal) of the cervical canal should b

reater than 14 millimeters. If the sagittal diameter is ss than 14 millimeters, the propensity for spinal cord mpression is present.

In addition, osteophytic projections may cause mpression of the vertebral artery in the vertebral anal and may result in symptoms of cerebrovascular nsufficiency involving the posterior circulation of the rain.

Neck pain associated with osteoarthritis of the ervical spine is not usually associated with neurologi- al changes. The neurological examination may be mpletely normal except for limitation of range of notion of the neck. In degenerative osteoarthritis, the eck pain may be chronic. It is important to realize hat x-ray changes of osteoarthritis may be seen in atients who are *asymptomatic* and *minor* x-ray hanges may be present in patients with severe ymptoms.

TREATMENT

The basic treatment of neck pain involves placing he neck at rest. The use of heat or ice or local nesthetics may yield temporary relief in myalgia and cal muscle spasm. A cervical collar will help to educe intervertebral movement and thus relieve neck nd arm discomfort for it is the intervertebral novement that tends to cause compression, irritation, nd stretching of the nerve roots. Prolonged use of a llar may produce muscle atrophy and stiffness and ead to a dependency syndrome.

Cervical traction will open the foramina which tends o decompress nerve roots and thus relieve radicular ain. Traction should be adjusted so that the neck is n a position of slight flexion for optimum comfort.

If patients do not respond to these conservative neasures, then myelography may be indicated espe- ially if neurological changes are detected. It is mportant to emphasize that many patients with steoarthritic changes of the cervical spine may resent with long tract findings indicative of my- lopathy in the *absence* of complaints of neck, houlder, or arm pain.

If the myelogram correlates with the patient's eurological findings, in a patient with radiculopathy, hen a surgical approach such as anterior diskectomy nd anterior interbody fusion may be appropriate. Occasionally a posterior spinal approach with fora- inotomy might be the surgical procedure of choice. If patient with myelopathy has a positive myelogram, hen a decompression procedure, either anterior or osterior, may be indicated.

ASE 1.

A 68-year-old female was admitted to the hospital ith a nine-month history of difficulty walking. She oted weakness in hands and legs and paresthesiae in he extremities. The patient was unable to ambulate.

A neurological examination revealed increased eflexes in the upper and lower extremities and a ensory level at C4. A myelogram revealed evidence of



Figure 1
A lateral myelographic film showing severe compromise of the cervical spinal cord compatible with a myelopathy secondary to osteoarthritic changes.

marked osteoarthritic changes compromising the cervical cord. (See Fig. 1)

A decompressive laminectomy and foraminotomy were accomplished at C3, C4, and C5. One month postoperatively the patient became ambulatory and has remained ambulatory during the past five years, although she continues to have residual evidence of long tract involvement.

RHEUMATOID ARTHRITIS

The cervical spine demonstrates rheumatoid changes in approximately 40% of rheumatoid arthritic patients. The synovial (apophyseal) joints and the bursa around the atlas and axis are involved by synovial proliferation and swelling. Formation of a pannus of granulation tissue with destruction of bone and ligaments results in instability in this area.

The atlantal axial joint is most often involved in rheumatoid arthritis and subluxation is most common at this level due to destruction of the transverse ligament of the odontoid. Subluxation may occur at other levels in the cervical spine as well.

The symptoms experienced by the patient will depend upon the sagittal diameter of the canal and the cervical vertebra affected. In patients who have rheumatoid involvement at C1-C2 with resultant subluxation, the patients may notice marked tingling and numbness in the extremities with forward flexion of the neck, (the so-called Lhermitte's sign which was originally described in patients with multiple sclerosis). These patients may also have symptoms and signs of a myelopathy. A lateral x-ray of the cervical



Figure 2
A lateral x-ray of the cervical spine showing subluxation of C1 on C2 with an increased distance between the anterior aspect of the adontoid process and the atlas.

spine taken in neutral position and one taken in forward flexion under strict medical supervision will yield the diagnosis.

TREATMENT

Treatment for a C1-C2 subluxation in rheumatoid arthritis is directed at minimizing the forward dislocation and may involve the use of external bracing with a cervical collar or a four-poster brace. Those patients who continue to have symptoms while in a brace or who are unable to tolerate bracing may require surgery. Rheumatoid arthritic patients whose subluxation does not reduce satisfactorily are candidates for an immobilization procedure consisting of posterior cervical wiring and fusion. Case 2 illustrates this condition.

Subluxations at other levels of the cervical spine are also treated by cervical fusion procedures.

CASE 2.

A 40-year-old male auto mechanic was admitted to the hospital with a 20-year history of rheumatoid arthritis. His recent complaints of pain in the posterior cervical area with radiation to the posterior occipital area led to a cervical spine x-ray which demonstrated subluxation of C1-C2. (See Fig. 2) Satisfactory reduction of the subluxation could not be achieved with a four-poster brace.

A posterior wiring and fusion of C1, C2, C3 was accomplished. Post-operatively the patient was symptom free.



Figure 3
A tomographic section showing invagination of the odontoi process into the foramen magnum.

A condition called pseudobasilar invagination ma occur in which there is herniation of the odontoi process into the foramen magnum. The tip of th odontoid process is not normally more than 4.5 mm above a line drawn from the hard palate to the occiput (Fig. 3) These patients will suffer from effects o compression of the medulla oblongata and ma present with ataxia, nasal speech, regurgitation o food and liquid, and eventually death.[3] The upwar herniation of the odontoid peg through the forame magnum in rheumatoid arthritics is the result o destruction of the lateral atlanto-axial joints.

A surgical decompression procedure in which th rim of the foramen magnum and the lamina of th upper cervical vertebra are removed, allows th medula oblongata to glide posteriorly away from th odontoid process. A more physiologic surgical pro cedure would be to resect the odontoid proces through a transoral approach. The latter approach because of risk of infection in these debilixate rheumatoid patients who are usually on steroids o bone marrow depressants carries greater morbidity.

Patients with C1-C2 subluxation or pseudobasila invagination may also complain of shock-like sensa tions over the posterior cervical and posterior occipita regions. Occasionally the description may be that of "crawling" feeling in those areas. These abnorma sensations occur secondary to pinching of the C2 root with abnormal movement of the vertebra. Posterio rhizotomies of the C2 roots may be accomplishe following the decompression procedures described fo pseudobasilar invagination for relief of these symp toms. In patients undergoing posterior fusion pro cedures for C1-C2 subluxation, these abnorma sensations are usually relieved following achievement of stabilization. Case 3 is illustrative of pseudobasila invagination.

CASE 3.

This 70-year-old female rheumatoid arthritic wa





igure 4
A] Lateral x-ray of cervical spine showing destruction and collapse of C7 vertebral body.

[B] Postoperative lateral x-ray demonstrating the insertion of an acrylic replacement for the resected C7 vertebral body.

admitted to the hospital because of severe neck pains and what she described as "a creeping" feeling over the back of her neck and head, as well as episodes of "blacking out" with flexion of her neck; she has had a history of rheumatoid arthritis for a period of 30 years with joint involvement.

Neurological examination revealed complaints of numbness in her hands with flexion of her neck, as well as paresthesiae in the posterior cervical region on neck flexion.

X-rays revealed evidence for pseudobasilar invagination. (See Fig. 3).

She underwent decompression of the foramen magnum and the arch of C1 with bilateral C2 rhizotomies. Postoperatively she was relieved of her previous symptoms referable to the cervical region and was discharged from the hospital one week following surgery.

TUMORS

Occasionally tumors involving the cervical spine will mimic the symptoms of arthritis of the neck and will cause symptoms of neck pain, radiculopathy, or myelopathy. Case 4 will illustrate the diagnostic and treatment measures.

CASE 4.

This 59-year-old patient presented with the symptom of neck pain for several months with the sudden onset of severe radicular pain radiating into the upper extremities. Cervical spine films revealed collapse of C7 and destruction of the pedicles. (See Fig. 4A). A myelogram revealed a block at C7.

An anterior decompression of the cervical spine with excision of the collapsed C7 vertebra and replacement with an acrylic prosthesis was accomplished. (See Fig. 4B). The patient was ambulatory and pain free the day following surgery. The pathological diagnosis was sarcoma. The patient expired six months following diagnosis with widespread metastatic disease, but was free of his previous neck and arm symptoms at the time of his death.

SUMMARY

Painful neck and arm symptoms are extremely common in medical practice and usually respond quite well to a conservative course of therapy. Those patients who do not respond to conservative therapy or who have long tract findings of a myelopathy deserve neurosurgical investigation. Arthritis patients may benefit from surgery to the cervical spine which includes cervical diskectomy, foraminotomy, decompressive laminectomy, or cervical fusion procedures.

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Modern Management of Gout

Eric P. Gall, M.D.

Gout, an ancient disease with a great deal of historical interest, is now easily diagnosed and treated. With modern techniques there is no longer any reason for patients to suffer repeated painful attacks of the acute arthritis associated with this disease. Joint destruction which may accompany chronic tophaceous gout should no longer occur. It is one of the few rheumatologic diseases in which a well defined diagnostic test exists. The treatment, though not curative, will prevent symptoms in a compliant patient.

The "pyramidal" approach to therapy of rheumatic disease was first popularized by Dr. Charley Smyth in the treatment of rheumatoid arthritis. In figure 1 this pyramid is adapted to the treatment of gout, the "gout house." For solidarity, the structure must have a strong foundation. Here the foundation requires definitive diagnosis, proper laboratory confirmation, and good patient education.

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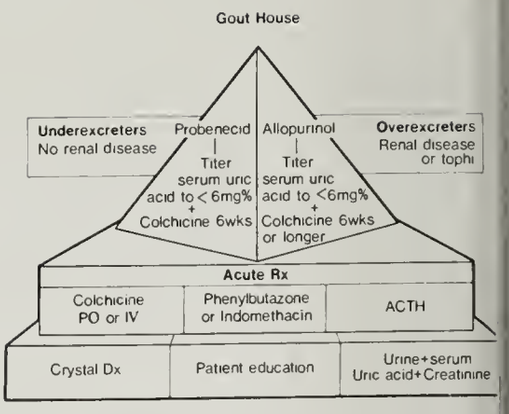


Figure 1

The definitive diagnosis of gout is established b demonstrating appropriate crystals in synovial fluid This is easily done in the physician's office by needl aspiration of the joint. A polarizing microscope with first order red compensator is requisite and can b inexpensively adapted by using polaroid film o material from polaroid sunglasses. (Figure 2). A recently described, the first order red compensato may be made by placing two strips of clear "Scotc Tape" on a microscope slide.[2]

The compensator may also be purchased commer cially. The polarizing lenses are placed on either side o the specimen so that the axes are perpendicula causing a dark background. (Figure 2). A birefringen crystal will now be readily apparent as a bright area.

The compensator must be placed between the tw polaroid plates giving a red background and th crystals blue and yellow color. Depending on the colo in relation to the axis of the red compensator, th crystals may now be identified definitively as sodiun

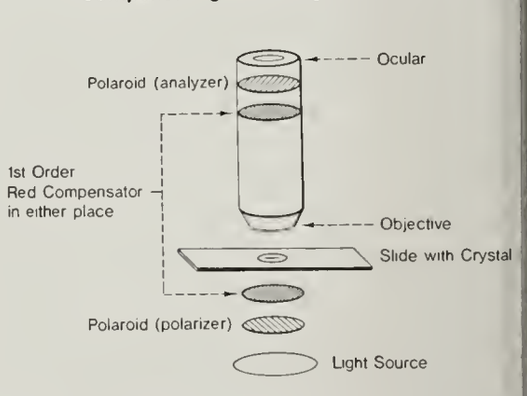


Figure 2

TABLE 1

	Calcium Pyrophosphate	Sodium Urate
Crystal Shape	Rhomboid	Needle
Birefringence	Weak	Bright
Characteristics of color in reference to the Axis of Red Compensator	Blue-Parallel Yellow-Perpendi'r	Yellow-Parallel Blue-Perpendi'r

urate or calcium pyrophosphate. (Table 1). The reader is referred to a more detailed description of how this is done.[1]

If crystal identification is not possible, a probable diagnosis of gout is suggested by this triad:

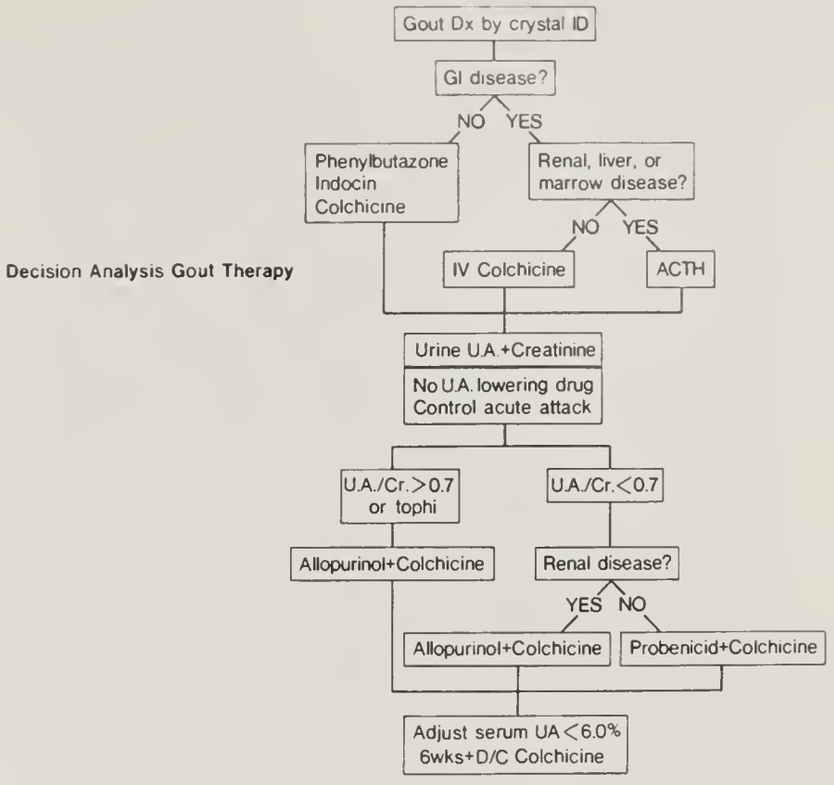
1. Acute monarticular arthritis
2. Elevated serum uric acid
3. Response to colchicine within 24 hours

There are, however, pitfalls in such a tentative diagnostic approach since as many as 30 to 50% of patients with gout have normal uric acid levels during their acute attacks. Moreover, other forms of arthritis may occasionally respond to colchicine and certain patients with polyarticular gout mimic rheumatoid arthritis. Thus definitive diagnosis requires crystal demonstration.

Once a definitive diagnosis has been made, a treatment program should be set up. The first level of the "gout house" (Figure 1) is that concerned with acute therapy. Figure 3 illustrates a decision analysis for treatment. In the absence of peptic ulcer, phenylbutazone, indomethicin or colchicine are administered orally. The first two drugs are preferable since they work rapidly, usually without inducing GI toxicity, a characteristic of colchicine. The latter drug is reserved for diagnostic use when crystal identification is impossible. Treatment of the acute attack usually requires 48 to 72 hours until the symptoms subside. Recommended doses are: phenylbutazone, 200 mgs q.i.d. with meals; indomethicin, 50 mgs q.i.d. with meals; colchicine, 0.5 mgs every hour until relief of symptoms, nausea or diarrhea appear or a total dose of 6 mgs has been given. In the event of gastrointestinal ulcer intravenous colchicine is well tolerated at a dose of 2 or 3 mgs diluted in 5% dextrose and dripped in slowly over 30 minutes. Additional 0.5 mgs amounts may be given if the attack does not subside; the aggregate amount should not exceed 4 mg. Alimentary tract disturbance is rare when colchicine is given intravenously. It is metabolized by liver and renal routes and since significant marrow toxicity may rarely occur, IV colchicine should not be used in patients with significant disease of the marrow, kidney or liver. ACTH gel may be used in these situations at a dose of 40 to 80 units I.M. At this point it is inappropriate to lower serum uric acid for two reasons.

TABLE 2

	24 hour Urine Uric Acid	Urine Uric Acid/Creatinine RATIO
Over-Producer	More than 800mg/24 hrs	More than 0.7
Under-Excreter	Less than 600mg/24 hrs	Less than 0.7



Decision Analysis Gout Therapy

Figure 3

TABLE 3

Common Causes of Gout

Over-Producer	Under-Excreter
Enzyme Defect (Genetic)	A. Intrinsic Renal Disease
Myeloproliferative Disorder	Renal Failure
	Lead Toxicity
Psoriasis	B. Competion for secretion
Cytotoxic Drugs	Drugs (Diuretics,
Hemolytic Disorders	Radiographic dyes, etc.)
	C. Idiopathic Gout

A rapid reduction before the termination of the acute episode causes prolongation of the attack. In addition, 24 hour urine and serum studies are affected by such therapeutic maneuvers.

Serum and urinary uric acid (U.A.) and creatinines (Cr.) provide additional important information which allow the clinician to separate the individual who overproduces uric acid from those individuals who underexcrete. (Table 2). Once the patient is categorized, other diagnostic considerations should be pursued. (Table 3). Overproducers may have malignant diseases or genetic predisposition to gout. In underexcretors one must look for underlying renal disease, lead toxicity, a variety of drugs and metabolic disorders.

Only when the acute attack has fully subsided should treatment for hyperuricemia begin. This appears as the upper floor of the "gout house" in

figure 1. All patients should receive a maintenance dose of colchicine 0.5 mgs b.i.d. or t.i.d. until 6 weeks after all acute symptoms have resolved or until the tophi have disappeared. There is evidence that maintaining colchicine treatment during this period reduces the number of recurrent gouty attacks.[3] All "over-producers" are given allopurinol as are patients with renal impairment. Those with adequate renal function and who are under-excretors may be given probenecid which is a slightly less toxic drug. Proper treatment with either of these uric acid lowering drugs requires titration of the serum uric acid to levels below the solubility product of this metabolite in the serum, i.e. 6 mgs% or lower. Allopurinol is usually begun at a doseage of 300 mgs per day and may be taken once daily. Probenecid is usually begun in doses of 500 mg b.i.d. Each of these drugs may be raised at weekly intervals (allopurinol 100 mgs at a time, probenecid 500 mgs at a time) until proper serum uric acid levels are attained. Prevention of serum uric acid elevation should be a life long program in gout.

The treatment of asymptomatic hyperuricemia is a controversial area. The guidelines which are recommended include serum uric acid levels which rise above 10 mg % in individuals with urinary uric acids greater than 800 mg/day. These individuals have an increased risk of urinary calculi and are treated for this reason, usually with allopurinol.[4] Otherwise most patients who have elevated serum uric acids are not prophylactically treated unless they rise to alarming levels of 20 mg % or higher, or until they become symptomatic.

Patients properly managed in the above manner should lead a symptom free existence from gout and should never develop destructive urate arthropathy. Proper diagnostic procedures will differentiate gout from rheumatoid arthritis and pseudo gout, disorders with which it is frequently confused. Patient education will help assure compliance to the life long treatment which prevents complications of this common disorder.

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Evaluation of the Child with Monoarthritis

Clifford DeBenedetti, M.D.

The child with arthritis involving a single joint can represent a process with innumerable possibilities. The purpose of this article is to present an approach to the child with monoarthritis that most often will provide either a rapid diagnosis or eliminates possible problems leading to early joint destruction.

The clinician should look on monoarthritis as an emergency requiring early diagnosis and institution of appropriate therapy. Delay in diagnosis, especially when dealing with an infection, can have lifelong consequences with future joint dysfunction. Once infection and hematologic disorders have been ruled out, a time of watchful waiting may be necessary before a specific diagnosis can be made.

HISTORY

A complete history with thorough review of systems can do much to insure an appropriate diagnosis. Recording basic information concerning the condition

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will be very helpful; i.e., joint involved, duration, persistent or intermittent swelling, etc. Although trauma is a leading cause of monoarthritis, it is invariably included in the history of many non-traumatic problems in children. Given the frequency of falls in children, it would be unusual not to have a history of trauma in a patient with a single swollen knee. Parents will often relate the onset of rheumatic disorders to an injury. In addition, a history of trauma with penetrating injuries is extremely important. Patients have developed septic arthritis after falling on pins projecting from carpeting. Patients who sustain direct joint penetration with plant thorns may develop a chronic synovitis which can be very difficult to distinguish from infection or JRA.[1]

Children with septic arthritis will usually have a prodrome involving upper respiratory signs and symptoms. From birth through the first month of age, staphylococcus aureus and gram negative bacilli are the most common causes of septic arthritis. Between four months and eighteen months of age haemophilus influenze type B is by far the leading cause of septic arthritis. After two years of age staphylococcus aureus again becomes the most common cause. Gonococcal arthritis is seen in all age groups, but is becoming especially prevalent in teen-agers; therefore, a detailed sexual history is necessary. The duration of disease activity must be estimated. For example, tuberculous and fungal infections may smolder along for months as compared to staphylococcal or haemophilus influenzae, which have acute onset.

Monoarthritis can be a manifestation of systemic disease and can precede the onset of overt disease. For example, children have been reported to have the onset of monoarthritis weeks to months before the primary disease asserts itself. This is seen with regional enteritis, ulcerative colitis, leukemias and neuroblastomas, so that detailed questions should be addressed as to appetite, malaise, vague abdominal pain, weight loss, recent growth retardation, presence or absence of intermittent fevers, stooling pattern, bleeding diathesis, etc. The patient with severe hemophilia should be well known to you by the time he presents with a hemarthrosis. A history of recent immunizations in an adolescent female with arthritis is important since rubella vaccine may be followed by monoarthritis; usually the knees, two to eight weeks post-injection.[2] The family history is not helpful in children with juvenile rheumatoid arthritis. There does not seem to be a greater incidence of rheumatoid disease in families of children having rheumatoid arthritis than the general population. However, the presence of ankylosing spondylitis in the family history can be very helpful. It is estimated that 20% of the patients with ankylosing spondylitis develop their disease as teen-agers with a peripheral arthritis, usually involving the hips or knees.[3]

PHYSICAL EXAMINATION

Don't miss the whole by looking only at the part. A complete physical (head to toes) with the patient undressed is mandatory. Try not to concentrate on the involved joint until a careful exam of other systems is completed. Often clues can be found which will facilitate an accurate diagnosis, i.e., iritis, hepatosplenomegaly, general adenopathy, pleural or pericardial friction rubs, skin rashes, muscle weakness or atrophy and restriction of motion of other joints. When examining an inflamed joint accurate recording of erythema, heat, swelling, tenderness and range of motion will be needed, and can be used in follow-up comparison examinations. Presence or absence of skin rashes may be very helpful in determining whether the process is JRA, various viral arthritides, systemic lupus erthematosus, or coccidioidomycosis. Attention should be paid to examination of the eyes, since frequently children with juvenile rheumatoid arthritis who have iridocyclitis will not have any complaints or physical findings referrable to the eye. For this reason, a slit lamp examination at some time in their evaluation should be done. Iridocyclitis occurs most frequently in children with pauciarticular onset of JRA. This is the type in which less than five joints are involved during the first six months of disease activity. The other two types of onset, system (fever, rash, pericarditis, anemia) and polyarticular (most like adult RA with frequent positive rheumatoid factor) can both have iritis, but much less frequently than pauciarticular JRA.

X-RAY

Radiographic studies are generally not helpful. The best yield will be in certain mechanical derangements such as osteochondritis dessicans, Perthes' disease, and slipped capital femoral epiphysis. In rheumatic disorders, soft tissue swelling is often the only finding early in the course of disease and may not show significant bone changes for months to years. Occasionally JRA patients may show findings of juxta-articular demineralization and periosteal new bone formation. Either of these may also be seen in patients with osteomyelitis. Joint scans with technetium pyrophosphatase are useful in detecting early synovitis or osteomyelitis.[4]

SYNOVIAL FLUID ASPIRATION

Once mechanical disorders have been ruled out (such as torn meniscus or osteochondritis) synovial fluid aspiration is in order. The child with a single hot joint almost always requires synovial fluid aspiration. A recent article has stated that synovial fluid analysis is as basic to the management of monoarthritis as is the blood pressure in hypertensive patients or the hemoglobin in an anemic patient.[5] If a sterile procedure is followed, complications are very rare. Aspirations should not be delayed, since early diagnosis of infection is a must. If the primary care

DIFFERENTIAL DIAGNOSIS OF MONOARTHRITIS

	AGE
TRAUMA	
Hemarthrosis	
Torn Meniscus	
INFECTION	
Bacterial	Greater than 1 mo —staphylococcus aureus
	3 to 24 mos —Haemophilus influenza
	Greater than 24 mo —staphylococcus aureus
Tuberculous	
Viral	Rubella
	Hepatitis B
	Mumps
Fungal	Coccidioidomycosis
COLLAGEN DISEASES	
Juvenile Rheumatoid Arthritis	Systemic
	Polyarticular
	Pauciarticular
Systemic Lupus Erythematosis	
Ankylosing Spondylitis	
Inflammatory Bowel Disease	
HEMATOLOGIC	
Hemophilia	
Leukemia	
Sickle cell anemia	
NEOPLASM	
Neuroblastoma	
SYNOVIAL DISORDERS	
Hemangioma	
Villanodular synovitis	
MECHANICAL DISORDERS	
Subluxation of patella	
Chondromalacia patella	
Osteochondritis dessicans	
Perthes' disease	
Slipped epiphysis	

physician does not feel comfortable aspirating any particular joint, immediate referral to the most qualified person should be done. Depending on the severity of the process and the age of the child synovial fluid studies may be limited by the volume of fluid that can be aspirated, but at the very least a culture can be done. If an adequate volume is obtained, color and clarity should be noted, mucin clot and glucose measured with simultaneous blood glucose, and a cell count performed. AFB, anaerobic and fungal cultures should be plated. A chocolate agar plate should be available for direct innoculation when gonococcal arthritis is suspected. Primary gout is extremely rare in children, but Lesch-Nyhan syndrome and secondary gout can occur. Since it takes only a drop of synovial fluid and a polarized microscope, an examination for crystals shouldn't be skipped. Rheumatoid factor tests on synovial fluid do not increase the possibility of diagnosing juvenile rheumatoid arthritis when the serum rheumatoid factor is negative. The white count may be useful in enabling us to fit the findings into various broad groups of inflammatory activity. Based on the number of white cells present, certain hints can be gathered as to what the basic underlying process can be.[6]

BLOOD TESTS

A complete blood count with differential and sed rate are first completed. This will help to rule out leukemia, although patients have been followed for weeks to months with monoarthritis (called juvenile rheumatoid arthritis) who have gone on to develop acute lymphocytic leukemia at later times.[7] Blood cultures will greatly increase the chance of detecting infection in patients who are suspected of having septic arthritis.[8] Rheumatoid factor tests are seldom positive or helpful in making a diagnosis. Overall, children with juvenile rheumatoid arthritis have approximately a 20% incidence of positive rheumatoid factor tests. This is directly age related, the older children tending to have more in the way of positive rheumatoid factor tests than younger children. Antinuclear antibody tests are important for both SLE and JRA. When the ANA is negative it virtually rules out systemic lupus erythematosus. It has also been found that the presence of a positive antinuclear antibody is seen in approximately 88% of children with pauciarticular JRA with iridocyclitis.[9] Presence of a positive ANA usually in a low titre can be a good tipoff that these children should have slit lamp examinations without delay. In children who have a hemarthrosis, coagulation tests are indicated. PPD and coccidioidin skin tests should always be applied. Cocci serology is especially important to determine in this area of the country. In certain children the HLA B-27 test can be very helpful, though not diagnostic. This tissue type antigen is seen in approximately 8% of the caucasian population but is greatly increased in patients who have various spondylo-arthropathies. These are the so-called rheumatoid variant diseases—ankylosing spondylitis, Reiter's syndrome, psoriatic anthritis and inflammatory bowel disease with arthritis. Though the presence of this test does not allow an accurate diagnosis to be made, it certainly does alert

one to the possibility that the monoarthritis may be an early manifestation of one of the rheumatoid variant diseases.

BIOPSY

Both open and needle biopsy are available for use in children. Recently it has been shown that needle biopsy using a Parker-Pearson needle on an ambulatory basis is possible. The children tolerated the procedure with only local anesthesia and were able to be back at full activity within 24 hours. Whichever method is used, sufficient material must be obtained. In the series reported by Dr. Jacobs on needle biopsy in children[10] almost one-quarter of the patients had insufficient material obtained for study. Biopsy is most helpful in attempting to rule out other processes in the evaluation of the child with probable juvenile rheumatoid arthritis. TB, cocci, pigmented villonodular synovitis, synovial tumors, all can be detected by synovial biopsy.

ARTHROSGRAPHY AND ARTHROSCOPY

Installation of contrast materials can be done in most joints and is most helpful in diagnosing mechanical disorders. Arthroscopy in the hands of the experienced person can be very helpful. Its application is limited to the knee joint at this time and requires general anesthesia. Synovial biopsy can also be performed at the same time.

SUMMARY

The child that presents with monoarthritis requires a careful and thorough evaluation. It should be handled with some urgency, at least until an infectious cause can be ruled out. Once infection has been ruled out by appropriate synovial fluid studies, repeated re-evaluations may be necessary before the true diagnosis can become apparent. Before we glibly attribute the diagnosis to juvenile rheumatoid arthritis, remember that the criteria necessitates a period of at least six weeks of active arthritis before attributing it to JRA.

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Infectious Arthritis

J. Steven Strong, M.D.

Of the many inflammatory conditions involving the joint, septic arthritis is the most rapidly progressive and destructive. Fortunately, with the advent of modern antimicrobial therapy and culture techniques, specific therapy is available for control and eradication of the condition.

The cardinal rule in infectious arthritis is awareness and recognition of such an inflammatory condition. In the differential diagnosis of oligo or monoartericular arthritis in both adults and children, septic arthritis must be considered as the number one suspect. Monoarticular and oligarticular arthritis includes gouty arthritis, pseudogout, psoriatic arthritis and juvenile rheumatoid arthritis in children. Infectious arthritis is the most rapidly progressive and therefore must be diagnosed during the first few hours after symptoms develop. Patients with impaired host defenses due to their primary disease or to therapy with corticosteroids or immunosuppressive drugs have an increased risk of infections by opportunistic pathogens. A history of chills, fever, migratory polyarthralgias followed by a persistent arthritis in one or two joints and an erythematous vesiculopustular rash are strongly suggestive of gonococcal arthritis.

From: Holbrook-Hill Medical Group, Ltd., Tucson, AZ.

When one suspects septic arthritis, a history documenting the general medical status of the patient and his predisposing conditions such as sexual activity, intravenous drug abuse, previous joint disease and current medications should be carefully obtained. Following a thorough physical examination, cultures of blood and of extra-articular sites of infection (including sputum, urine, and any other suspicious sites) should be obtained. Finally, the affected joint must be immediately aspirated.

With proper aspiration techniques, a working diagnosis of septic arthritis can usually be made. After disinfecting the skin with iodine and alcohol, the joint fluid may be collected in a heparinized syringe. A portion can be transferred to a sterile tube for bacterial, mycobacterial and fungal cultures. However, for immediate diagnostic purposes, the fluid should be gram and acid-fast stained. The remaining synovial fluid can be examined for total WBC and differential, crystals and glucose levels. It should be remembered that acetic acid should not be used as the diluent in the white cell pipette since acetic acid will agglutinate synovial protein. Finally, a polarizing light microscope is necessary for the identification of urate or calcium pyrophosphate crystals.

The information from such a synovial fluid analysis can be most useful in diagnosing septic arthritis. Total white blood counts greater than 50,000 with 95% polymorphonuclear leukocytes narrow the differential diagnosis to an infectious or gouty etiology. The lack of crystals under the polarizing lens will effectively eliminate gout or pseudogout. A positive gram stain occurs in between 50 and 80% of septic arthritis.

When a septic process involves the joint space, a specific sequence of events occurs. There is a migration of inflammatory cells to the point of infection. Since the joint is a closed space containing the infectious process, the fluid pressure is increased. With this comes slow egress from the joint permitting the accumulation of products of inflammation. Osteolytic and chondrolytic enzyme activity increases resulting in rapidly progressive destruction of cartilage and bone. It is imperative that drainage of purulent exudate should be carried out regularly. This includes adequate needle drainage of the involved joint at the time of the suspected diagnosis and once or twice a day after therapy has begun to prevent accumulation of destructive products of inflammation and to assess the response to therapy. This can be done by regular cell counts and differentials as well as appropriate cultures. Repeated radiologic evaluation of the joint at three to six day intervals also helps to assess the degree of bony destruction which may have occurred during the infectious process.

Therapy of a septic joint should begin immediately after the assessment of the general medical status and appropriate joint fluid findings. At the time of joint fluid analysis, gram stain may be extremely helpful in directing initial antibiotic therapy. (Please see Table #1 for description of gram stain, presumed bacteria and initial antibiotic therapy). Antibiotic therapy should be given intravenously. Synovial fluid concentrations of most antibiotics can be effectively achieved by intravenous administration except for large molecular weight drugs such as Amphotericin B, Polymyxins and Streptomycin. After appropriate identification by culture of the etiological agent, specific antibiotic intravenous therapy should be continued. (Table II). It must be remembered that certain etiological agents such as gonococcus, fungi, Mycobacterium and atypical Mycobacterium may be very difficult to culture from a single synovial fluid. Since these conditions are of a lower grade inflammatory characteristic, white counts may not approach as high a level and percentage of neutrophils may be less than 95%. In suspect cases, improvement of diagnostic accuracy to over 90% can be achieved by appropriate synovial tissue biopsy, culture and pathological review.

A final comment should be made in regard to drainage of involved joints. Peripheral joints can be adequately drained by needle aspiration on a daily basis as mentioned above. Because of the peculiar blood supply to the hips and rapidly progressing fluid pressure of the closed hip space, orthopedic consultation and immediate open drainage may be necessary. Peripheral joints which have been followed carefully with appropriate antibiotic coverage and have not responded to medical management over five to seven days again become candidates for orthopedic intervention.

TABLE I
STARTING ANTIBIOTIC THERAPY BASED ON GRAM STAIN OF SYNOVIAL FLUID

Gram Stain	Presumed Bacteria	Initial Antibiotics
Gram-negative intracellular diplocci	Gonoccoci	Penicillin G
Gram-negative pleomorphic rods	Haemophilus	Ampicillin (or Chloramphenicol
Gram-negative uniform rods	1. Pseudomonas	Carbenicillin and Amino Glycoside
	2. Coliform Organisms	
Gram-positive Cocci	1. Pneumococci	Penicillinase-resistant Penicillin*
	2. Streptococci	(Pending bacteriologic identification)
	3. Staphylococci	
No bacteria seen	1. Neonates: Staphylococcus or coliforms or Group B Streptococci.	Penicillinase-resistant Penicillin* plus Amino glycoside
	2. Infants: Haemophilus	Ampicillin (or Chloramphenicol)
	3. Older children and adults, Staphylococcus (Gonococci)	Penicillinase resistant Penicillin*
	4. Immunosuppressed patients	Penicillinase-resistant Penicillin* plus Amino glycoside

* Methicillin, Nafcillin, Oxacillin

TABLE II
PREFERRED ANTIMICROBIAL THERAPY
AND DURATION OF TREATMENT

tiologic Agent sual prevalence order	Drug	Minimum Duration* Of Treatment
1-Neisseria gonorrhea	Penicillin G	3-5 D
2-Staphylococcus aureus	Penicillinase-resistant penicillin**	3-4 W
3-Streptococcus (Diplococcus pneumoniae)	Penicillin G	10 D
4-Streptococcus pyogenes (Group A)	Penicillin G	10 D
5-Streptococcus agalactiae (Group B)	Penicillin G	10 D
6-Haemophilus influenza	Ampicillin (or Chloramphenicol)	10 D
7-Pseudomonas Aeruginosa	Carbenicillin (plus Amino glycoside)	6 W
7-Escherichia coli	Amino glycoside***	3-4 W
7-Klebsiella-Enterobacter sp.	Amino glycoside***	3-4 W
8-Neisseria meningitidis	Penicillin G	3-5 D
9-Bacteroides fragilis	Chloramphenicol (or Clindamycin)	(?)-2 W
9-Bacteroides, other sp.	Penicillin G	(?(-2 W
10-Clostridium	Penicillin G	(?)-2 W
11-Fungi	Amphotericin B (or Flucytosine)	6 W
11-Mycobacterium tuberculosis	Isoniazid (Rifampin) and Ethambutol (or PAS)	18 M 18 M
12-Salmonella sp.	Ampicillin (or Chloramphenicol)	3-4 W

*D - days; W - weeks; M - months
**Methicillin, Nafcillin, Oxacillin
***Specific sensitivities required

In these days of changing social habits and readily available antimicrobial therapy for uncomplicated minor infections, one must become more attuned to the times—ever suspicious of septic joint inflammation. The more promiscuous attitude of today's youth lead to the ever greater incidence of gonococcal arthritis. Previous exposure to antibiotic therapy leads to virulent strains of resistant microbial agents.

The septic joint is a challenge to diagnose and may be a rewarding experience to treat.

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How To Treat Rheumatoid Arthritis with Penicillamine

Harold C. Tretbar, M.D., FACP

It is difficult to treat a disease when you do not know what causes it and there is no known cure. For these reasons, rheumatoid arthritis has been treated with many different medications. Most of our standard drugs for rheumatoid have been found to work empirically. For instance, Gold salt injections were being used to treat tuberculosis when it was found to be quite effective in rheumatoid arthritis. Similarly, Penicillamine, originally designed to chelate excess copper in Wilson's disease, is becoming a primary drug in the treatment of rheumatoid arthritis.

Penicillamine is somewhat related in structure to penicillin. It is beta beta dimethylcysteine which is a sulfur containing amino acid. D-Penicillamine is rapidly absorbed through the intestinal wall with a peaked blood concentration at one hour after ingestation. It is oxidized by the cytochrome system and excreted as a disulfide.

It is not clear how Penicillamine reduces inflammation in rheumatoid disease. It can cause dissociation of the rheumatoid factor with reduction of the serum levels. However, it does not parallel improvement of the joints.[1]

Penicillamine has been demonstrated to interfere with cross linking of tropocollagen molecules. Theoretically it should decrease collagen production in scleroderma but it has been used clinically with only mild success.

In 1962 Jaffe began using Penicillamine for rheumatoid arthritis. He has been able to refine the dosage schedule so that it is generally well accepted by the patient.[2] Following reports of carefully controlled studies in 1973 and 1974, British rheumatologists have become avid supporters of its use. Their studies suggest improvement in 80% of the patients after 6-9 months of treatment.[3],[4] Their enthusiasm is now gaining support in the United States.

Unfortunately, as with many of our medicines for arthritis, side effects can still be a problem. Revised dosage schedules have avoided many of the unpleasant minor side effects. Major side effects are still possible

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but are less frequent.

I will try to summarize how to use Penicillamine in rheumatoid arthritis and refer the reader to key articles for detailed information.

INDICATIONS FOR USE: In general, progressive polyarticular rheumatoid seems to respond the best. There may be better improvement in those patients that are positive for rheumatoid factor. Extra articular involvement such as arteritis or neuritis has shown excellent response.[5] The general feeling is that Penicillamine should be considered as a second line drug; when you consider Gold or cytotoxic agents. It may be particularly helpful for those patients who are Gold failures.

CONTRAINDICATIONS: There apparently is no cross sensitivity in patients allergic to penicillin. It should not be used in the rheumatoid patient who has marked leukopenia (white cell count less than 2,000), thrombocytopenia (below 70,000), or proteinuria (greater than 1 gram per day).

TREATMENT SCHEDULE: It has been shown that a go slow, go low schedule avoids many side effects. Initially, Jaffe started his patients on 2-3 grams per day. Many of them developed loss of taste or smell. Buccal ulcerations were common. Indigestion was frequent and skin rashes were difficult problems. Jaffe has demonstrated that a schedule of 250 mgs. once a day for one month, then B.I.D. for one month and finally T.I.D. as a full dose, is well tolerated.[2]

Laboratory tests should include weekly CBC, platelets and urinary protein during the first month and then every 1-3 months after the patient is on long term therapy.

SIDE EFFECTS: Occasionally mild nausea or indigestion will develop but is temporary. Loss of taste will occasionally last for several weeks but will clear spontaneously. Buccal ulceration is uncommon but may occur after 6 months of treatment. It may respond to temporary decrease of the dosage.

Two types of rash may be encountered. A rash early in the course of treatment may appear like an ampicillin rash and clears rapidly after discountinuing the drug. Frequently the drug may be restarted without re-occurrence. The late rash may be an intense pruritus. Bullous lesions can develop or they can be discrete scaly macules. Occasionally these may require discontinuation of Penicillamine.

Adverse effects may be found in the blood count. Thrombocytopenia may be the most difficult to detect as there may be a rapid onset occuring after several months of treatment. Thrombocytopenia usually corrects itself when the medication is stopped. It should be discontinued if the platelet count is below 70,000.

Proteinuria is of considerable concern. Minor proteinuria, up to 2 grams a day, can be accepted. Larger amounts usually indicate an immune complex deposition in the kidneys. Occasionally nephrotic syndrome may develop. It is wise to decrease the dosage if proteinuria is less than 2 grams per 24 hours and discontinue it if above 2 grams. The urine should continue to be monitored carefully.[6]



Figure 1
Chronic lower leg ulcers of rheumatoid vasculitis before treatment

Other unusual side effects to Penicillamine have been recorded. They include syndromes similar to Lupus Erythematosis, myasthenia gravis and polymyositis. Goodpasture's syndrome has also been reported.[7] These have cleared after discontinuation of Penicillamine.

INCIDENCE OF SIDE EFFECTS: Hill has followed 180 patients up to 3 years. After 6 months of use 8% were withdrawn from the study because of adverse reactions (mostly thrombocytopenia). At 12 months 30% had to withdraw because of rash or proteinuria. By 18-24 months another 5% had significant proteinuria.[6]

COMPARISON STUDIES: Penicillamine has been compared to both Gold salts and azathioprine in single blind studies. After one year of comparing azathioprine and Penicillamine in 65 patients, both were equal in efficacy and toxicity. Eighty-six patients were studied to compare Gold and Penicillamine. Both were equally effective but more were withdrawn because of Gold side effects.[4]

LONG TERM USE: Reduction in joint inflammation may be slow when using Penicillamine. Maximum improvement may take 6-9 months. Other anti-inflammatory agents can be continued while on Penicillamine. These include anti-malarials, non-



'igure 2
.eft leg after one month of Penicillamine.



Figure 3
The ulcer of the right leg has healed. Marked improvement of left leg after 7 months of treatment.

steroidal anti-inflammatories, aspirin and steroids. The patients with rheumatoid arthritis who do well on Penicillamine may continue to be treated for years while trying to reduce the dosage to 250 mgs. to 500 mgs. a day.

CASE REPORT: Patient, M.G., age 61 developed rheumatoid arthritis at age 27. Her polyarticular sero-negative arthritis had been treated with various medications including Gold in the late 1940's. She has been on steroids since 1950.

She first developed ulceration of the lateral side of the left foot from rheumatoid vasculitis in 1967. This healed without difficulty. In 1972 she then developed similar vascular ulcers over the medial sides of both feet. These were treated symptomatically with somewhat slow healing. In mid-1974 she again had the onset of extensive ulcerations of the left lower leg and ankle. They were skin grafted in April, 1975 and remained covered until the graft broke down in October, 1975. The ulcer of the left lower leg gradually increased in size and became more painful. In June, 1976 a new ulcer appeared on the right medial ankle. The ulcers were unresponsive to antibiotic ointment, Biozyme and compressive dressings.

I first saw the patient in September, 1976 with extremely painful ulcers as shown in photograph #1. Her blood count was normal. The Westergren sed rate was 46 mm/hr. The RA latex test was positive in a dilution of 1:5120. The ANA titer was positive at a dilution of 1:20 with a speckled pattern. She was started on Penicillamine 250 mgs. at bedtime. Previous medications including Aristocort 2 mgs. a day, 8 Bufferin a day and Plaquenil 250 mgs a day were continued unchanged. She tolerated the Penicillamine well and it was increased to twice a day 3 weeks later. She was then increased to 250 mgs. 3 times a day. She has had no complaints or side effects. The blood count and urinalysis remain normal. By November, 1976 the sed rate had decreased to 12 mm/hr. Westergren and in April, 1977 the sed rate was 8. The RA titer dropped to 1:2560.

Photograph #2 was taken in October, 1976 and photograph #3 in April, 1977. They demonstrate the slow but progressive healing of otherwise intractable ulcers. All pain has disappeared. The patient has no active synovitis and remains under further outpatient treatment.

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U of A
Grand Rounds

Sjogren's Syndrome: A Clinical and Immunologic Review

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Sjogren's Syndrome (SS) was defined by Sjogren in 1933[1] as a clinical entity including decreased secretion of lacrimal, salivary, and upper airway glands. There was occasionally parotid enlargement; arthritis occurred in most patients; and there was often an elevated erythrocyte sedimentation rate and anemia. The diagnosis could be confirmed by the Schirmer tear test or by staining of the conjunctiva and cornea with rose bengal.

Diagnostic criteria employed by most workers today are those shown in TABLE 1.

PROMINENT CLINICAL COMPLAINTS: Arthritis or arthralgia—Shearn's classic monograph on SS[2] reported a series of 80 patients. The most frequent first complaint in this series (occurring in 40%) was arthritis or arthralgia.

Early reports concerning the arthritis associated with SS are confusing. These patients had an unspecified sort of polyarthritis (degenerative joint disease, gout, rheumatoid arthritis [RA], connective tissue disorders other than RA, or only non-specific causes for joint symptoms). It was not until 1949 that a particular association with *rheumatoid* arthritis was indicated when increased rose bengal stainability in the eyes of patients with various rheumatic disorders, but most frequently RA, was noted.[3]

TABLE 1
GENERAL ASPECTS

Definite	
Sjogren's syndrome:	
The presence of *either*	1) Objective evidence of keratoconjunctiv's sicc
or	2) Characteristic patholog features in the lacrimal or salivary glands
Probable	
Sjogren's syndrome:	
The presence of *two of three* features:	1) Recurrent or chronic "idiopathic" salivary gland swelling
	2) Unexplained xerostomi
	3) Connective tissue disease

Shearn, M.: Sjogren's Syndrome. Philadelphia: W.B. Saunder Company, 1971, p. 11.

Promulgation of the American Rheumatism Asocia tion criteria for the diagnosis of RA and application o these criteria to patients with SS has permitted a clearer realization that approximately 50% of al patients with SS do have definite or probable RA.

Not all patients with SS and joint complaints hav RA. As can be seen in TABLE 2, the association of S with other disorders causing arthralgia or frank arthritis is relatively high.

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TABLE 2
ASSOCIATED DISORDERS IN 80 PATIENTS WITH SJOGREN'S SYNDROME

	No. of Patients*	Per Cent
heumatoid arthritis	24	30
aynaud's phenomenon	17	21
hronic pulmonary fibrosis	12	15
nexplained arthralgia	8	10
hronic hepatobiliary disease	5	6
ystemic lupus erythematosus	4	5
cleroderma	4	5
urpura hyperglobulinemia	4	5
hronic thyroiditis	3	4
yopathy	2	3
ranial arteritis	1	1

Some patients had more than one associated rheumatic disease.

hearn, M.: Sjogren's Syndrome. Philadelphia: W.B. Saunders ompany, 1971, p. 105.

Eye Complaints—Approximately 90% of patients ith SS have keratoconjunctivitis sicca (KCS) some me during their course (TABLE 3). Accessory and ajor lacrimal glands undergo progressive atrophy id destruction of the secretory epithelium, associated ith massive infiltrates of lymphocytes and plasma lls. With severe atrophy of the secretory apparatus, essication of the cornea and conjunctiva may result. lthough occasional patients with evidence of occular volvement (by biomicroscopy or lacrimal gland opsy) have normal *quantity* of tearing by the chirmer filter paper test, this remains the standard reening test.

Usually the onset of eye symptoms is insidious and ily in general correlates with the degree of lack of ar production. Frequent complaints from one series e shown in TABLE 4.

TABLE 3
GENERAL ASPECTS

Major Features of Sjogren's Syndrome In 80 Patients

	Number	Per Cent
Keratoconjunctivitis sicca	69	87
Xerostomia	52	66
Connective tissue disease	49	62
Abnormal salivary or lacrimal gland	29	37
Positive salivary gland biopsy	25	31

Shearn, M.: Sjogren's Syndrome. Philadelphia: W.B. Saunders Company, 1971, p. 12.

Oral Complaints—Atrophy of the accessory and major salivary glands causes a decrease in the rate of salivary secretion and subsequently a dryness of all areas that depend upon those glands for lubrication. This results in xerostomia in approximately two thirds of patients (TABLE 3). Most attention has been focused on the parotid glands although all salivary glands may be involved. The increased attention given parotid enlargement is possibly due to its being the most frequently obvious: Of the approximately one-third of patients with SS who have salivary gland enlargement, about 90% have enlargement of the parotids, whereas about 20% have enlargement of the submandibular glands.[2]

Biopsy of an involved gland would be the most direct way to diagnose salivary gland involvement. Although parotid or submandibular glands may be biopsied, the accessory salivary glands of the lip are more accessible and biopsy of these glands is accompanied by fewer complications than is biopsy of the major glands.

The technique of labial salivary gland biopsy has proved an important means of quantitating disease. Using a "focus score" based on the number of lymphocytes and plasma cells per 4 mm^2 of tissue,

TABLE 4
FREQUENCY OF SYMPTOMS

Presenting Symptoms	Frequency (percent)
Burning and smarting	16
Scratching and foreign body sensation	13
Excess of secretion	12
Dryness	11
Redness	9
Photosensitivity	6
Itching	5
Inability to cry or lack of tears	3
Diminished vision	1
Ulcers	1
Tiring, irritability, hurting, soreness, pain	23

Henderson, J.W.: Keratoconjunctivitis sicca. A review with a survey of 121 additional cases. Am J Ophthalmol 33:197-223, 1950.

Greenspan et al.[4] examined labial biopsies from 75 patients. Clinically mild cases had fewer foci and a higher percentage of plasma cells in these foci; severe cases had a greater number of foci and a higher percentage of lymphocytes.

Salivary flow measurements document the degree of hyposalivation. Whaley et al.[5] have found lemon juice stimulated salivary flow rates a sensitive method of assessing salivary gland function. Sialography is usually abnormal but has complications (e.g., acute parotitis) and necessitates a trained person to catheterize the duct. Radioisotopic scanning has recently been frequently mentioned as an important diagnostic maneuver. Sequential salivary scintigraphy (to study the uptake, concentration, and excretion of Tc^{99m} pertechnetate by the salivary glands) correlates well with the degree of clinical symptoms, with abnormality of salivary flow rate, and with abnormality in the sialogram. This scintigraphic procedure has proved in some hands useful to follow response to therapy in patients treated.[6]

No matter how diagnosed, the major clinical effec of salivary gland atrophy is dry mouth, or xerostomia As the salivary glands progressively atrophy, the rat of salivary flow goes from normal to lacking only i response to food or olfactory stimuli to complete spontaneous dryness.

At this point, the patient may have burning pai and paresthesias of the mouth and tongue; fissuring o the tongue, lips, and corners of the mouth; an bleeding. Speech becomes difficult due to dryness Patients may complain of dry, sore throat an hoarseness. Dry foods become difficult to swallow Stagnant food debris causes malodorous breath. Lac of saliva contributes to dental caries and may alte oral flora such that fungal organisms—particularl *Candida albicans*—flourish.

Although other disorders or even drugs ma produce xerostomia, the likelihood of SS being presen in a patient who complains of dry mouth is sufficientl high to cause Shearn to suggest[2] that idiopathic chronic xerostomia, especially in a middle-aged o older woman, should strongly suggest SS.

EXTRASALIVARY LYMPHOID ABNORMALITIES: In 1967, Talal et al.[7] reported eight patient with SS and extrasalivary lymphoid abnormalities. Ir all, lymphadenopathy or other lymphoid infiltrate (for example, of the lung or kidney) were sufficient t arouse a suspicion of lymphoma. Two patients ha macroglobulinemia, one had reticulum cell sarcoma and five had what was termed "pseudolymphoma' (node biopsies revealed complete destruction of noda architecture by an inflammatory, but not clearly malignant, cellular infiltrate).

They summarized 13 similar patients theretofor reported and commented specifically on the universa presence of salivary gland enlargement; high incidence of lymphadenopathy, splenomegaly, Raynaud's phenomenon; and relative infrequency of arthritis (which if present was not typically rheumatoid) (TABLE 5).

Anderson and Talal[8] put pseudolymphoma into perspective noting that lymphoproliferation in SS exists as a spectrum from merely infiltrated glandular tissues on one end through pseudolymphoma to frank

mphoreticular malignancy on the other end. They autioned that the prognosis in pseudolymphoma atients is guarded: Of six pseudolymphoma patients eported in 1967 five were dead by 1971 (two of eticulum cell sarcoma). They suggested early treatnent for pseudolymphoma patients to diminish or at east arrest the lymphoproliferative process and to ead to symptomatic improvement and to potential rolongation of life.

T and B Lymphocytes in SS—Cell-mediated immunity has been shown to be impaired in patients with S. Leventhal,[9] using phytohemaglutinin and streptolysin O as mitogenic agents, showed that lymphocytes in the majority of patients with SS were unctionally abnormal in their response to these gents. Nearly half of his patients, however, fell vithin the range of normal controls. Further, whereas 4% of normal persons demonstrated delayed hypersensitivity to dinitrochlorobenzene (DNCB) only 35% f the patients with SS developed this hypersensiivity. Other workers have reported higher perentages of patients with SS who responded normally o DNCB[10] but the number remains lower than the ercentage of normal controls who respond.

Talal et al.[11] looked at actual numbers of T ymphocytes in 20 patients with definite SS and four vith probable SS. The majority of patients had a ormal percentage of peripheral T cells. However, 9% had an abnormally low percentage of T cells by n immuno-fluorescent method and 17% had a lecreased percentage of T cells by rosette determinaion.

Other examples of altered immunologic reactivity

1. Drug allergy—Bloch et al.[12] called attention to a 3% incidence of drug hypersensitivity or allergy in atients with SS. Later workers[13] have looked pecifically at penicillin allergy and found that 17% of atients with RA are allergic to penicillin (not tatistically different from controls). However, from 4% to 41% of patients with RA plus SS are penicillin llergic.[13, 14] Whaley et al.[14] have suggested that any atient with RA who develops drug allergy should be screened for SS. Mixed usage of the terms "drug hypersensitivity"and "drug alergy" has confused interpretation of some reports. However, it seems reasonable for the clinician to keep in mind a potentially increased risk of drug reaction in patients with SS.

TABLE 5
RELATIVE FREQUENCY OF CERTAIN FEATURES IN SJOGREN'S SYNDROME WITH AND WITHOUT EXTRASALIVARY LYMPHOID ABNORMALITIES

Clinical Feature	With (%)	Without (%)
Female	85	95
Mean age (yr.)	58	49
Xerostomia	100	88
Keratoconjunctivitis sicca	82	89
Salivary gland enlargement	100	50
Arthritis	31	52
Lymphadenopathy	77	0
Splenomegaly	77	23
Leukopenia	77	26
Purpura	62	4
Vasculitis	54	22
Neuropathy	31	2
Raynaud's phenomenon	31	23

Talal, N.; Sokoloff, L.; Barth, W.F.: Extrasalivary lymphoid abnormalities in Sjogren's syndrome (reticulum cell sarcoma, "pseudolymphoma," macroglobulinemia). Am J Med 43: 50-65, 1967.

2. Serum Protein Abnormalities—SS is frequently accompanied by a diffuse hypergammaglobulinemia (TABLE 6). The highest average and peak gammaglobulin concentration occur in patients with SS *without* RA (these patients having a mean value of 2.5 g%).[12] In addition, non-specific inflammatory elevation of alpha-2 globulin and depression of serum albumin may occur. Isolated macroglobulinemia has been reported and hypogammaglobulinemia has been seen in a patient with SS and reticulum cell sarcoma.

TABLE 6
LABORATORY FINDINGS IN EIGHTY PATIENTS WITH SJOGREN'S SYNDROME

Laboratory Finding	Tested (No.)	Abnormal (No.)	(%)
Immunologic Abnormalities			
Rheumatoid factor	70	51	73
Hypergammaglobulinemia (1.4 g/100 ml)	71	40	56
Antinuclear antibodies	54	26	48
LE cell	53	9	17
False positive serologic test	80	4	5

Shearn, M.: Sjogren's Syndrome. Philadelphia: W.B. Saunders Co., 1971, p. 177.

Not only are *serum* gammaglobulins abnormal, local salivary gland immunoglobulin and rheumatoid factor (RF) synthesis are abnormal. IgA is synthesized locally in normal or slightly higher than normal amounts; IgG and particularly IgM are synthesized in significantly higher amounts.[6] Anderson et al.[15] showed decreased production of IgM, IgG, or RF in all patients who were begun on therapy with cyclophosphamide, prednisone, or x-ray after the initial biopsy and who had a repeat biopsy seven to 24 months later. Of patients who did not have new therapy begun during the interval between biopsies, 43% showed *increase* in at least one of the parameters measured and only one patient (14%) showed decrease.

Although serum viscosity has been known to correlate with gammaglobulin concentration, the titer of RF, and the presence of macroglobulin it was not until 1973 that documentation of the hyperviscosity syndrome in SS was made. Pruzanski[16] summarized nine reports of the hyperviscosity syndrome in connective tissue disorders. Five of these nine had SS. He noted that all had prominent hypergammaglobulinemia and very high titers of RF. He suggested that high levels of gammaglobulin and high titers of RF in patients with polyarthritis and multisystem involvement should raise the consideration of possible concommitant hyperviscosity state.

Recent interest has been shown in the beta-2 microglobulin: A low molecular weight, cell-membrane protein structurally related to immunoglobulin and present in low concentration in normal serum and urine. Talal et al.[17] showed that salivary beta-2 microglobulin concentrations were markedly increased in 30 patients with SS. By folloing beta-2 microglobulin concentration in patients on therapy they suggested that levels increased and decreased with exacerbations and effective therapy of the disease.

Michalski et al.[18] documented the same findings in a larger group of SS patients and correlated the levels closely with the degree of inflammation seen on labial biopsy. They further looked at *serum* beta-2 microglobulin concentration and showed that the highest concentrations occurred in those patients with renal abnormalities or extra-salivary lymphoproliferative complications.

3. Antibodies to Cellular Components—Antinuclear antibodies have been reported in one-half to two-thirds of patients with SS. No one pattern of immunofluorescence has proved to be diagnostic.

Alspaugh et al.[19] have studied precipitating antibodies in SS to antigens extracted from a human diploid lymphoid cell culture line. They have described three antibodies (SS-A, SS-B, and rheumatoid arthritis precipitin [RAP]). They showed that of all patients positive for SS-A, 94% had SS alone and of all positive for SS-B, 93% had SS alone. Conversely, of all patients positive for RAP, 89% had RA, with or without SS. Further, of all SS patients, only 5% were

ith RA alone, no patients were positive for SS-A or S-B). Perhaps more importantly, of 166 patients with LE, discoid lupus, scleroderma or mixed connective ositive for RAP and of patients with SS and RA, nly 12% were positive for SS-A or SS-B. (Of patients ssue disease only one was positive for SS-B and one as positive for RAP. This suggests a new system of ntigens nearly totally distinctive to the RA/SS mplex and not found in other connective tissue isorders.

Further amplification of the immunologic difference etween patients with SS alone and those with SS plus A has come from histocompatability antigen studies. n increased incidence of HLA-B8 antigen has been oted in SS. Chused et al.[20] have studied the D-locus eterminant, Dw3, known to be in linkage dysequibrium with the B8 antigen. They showed that Dw3 ntigen is even more closely associated with SS and hat, although 84% of patients with SS alone are Dw3 ositive, patients with RA plus SS have no increased requency of Dw3 antigen.

SUMMARY: What was once looked on as a benign lisorder of lacrimal and salivary glands has come to be een as a condition affecting many organs of the ody—sometimes with lethal effect.

SS is now regarded as a disorder of lymphoproliferaion and immunologic imbalance. There may be a uantitative or qualitative deficiency of T cells and B ells may proliferate, making aberrant immune esponses which we detect as autoantibodies and as ome of the clinical manifestations discussed. Frank ymphoreticular malignancy may result. Although RA loes occur in approximately 50% of patients with SS, he other 50% appear to be genetically and immunoogically different and at higher risk for certain clinical nd laboratory abnormalities. Although immunosupressive therapy of uncomplicated SS is not recommended, current understanding may allow us to dentify patients at high risk for complications (such s lymphoma, macroglobulinemia, or hyperviscosity syndrome) and early therapy of these patients may make them more comfortable and allow them to avoid hose complications later.

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An Ounce Of Prevention

On February 28, 1976 a new Professional Liability Act went into effect in Arizona followed on April 1, 1976 by the inception of Professional Liability Insurance coverage by the Mutual Insurance Company of Arizona. As a result of these occurrences, a number of things have developed in the malpractice arena in Arizona.

The Underwriting Review Committee of ARMA has handled a tremendous number of insurance applications, both original and re-insurance. Many lessons have been learned by those doing the reviews, some of which have hopefully been discreetly carried back to the local level. This committee has developed a special subcommittee that is available to physicians with special or recurring problems.

The Claims Review Committee of ARMA is gradually hearing more cases with the ultimate decision being made to either defend or settle the claim. In this committee also certain things became evident which might have been helpful in preventing the action.

MICA, itself, has developed a claims prevention program. One of the main thrusts of this program is to educate the physicians. Sometime ago, a letter was sent out over Dr. Brook's signature along with a monograph describing what to do when the Summons and Complaint are served. Many things can be done, however, before that missile arrives.

Among the advice being offered by the insurance people to those who wish to avoid being served or becoming embroiled in a costly claim are the following pointers:

—medical records are always a crucial part of the defense of any malpractice law suit. The more complete the records are, the easier it is to demonstrate that a physician has provided appropriate treatment. Notations should always be made when a patient refuses recommended treatment or hospitalization, or fails to follow medical advice. A brief note concerning discussions of the risks of treatment or surgery serves to counter claims that the patient did not give his informed consent.

—when a patient consistently jeopardizes his own health by failing to heed the advice of his physician, he places that physician at risk by forcing the physician to provide less than optimal care. Often, the only solution is to discharge the patient from your care But this must be done in prope fashion. Your malpractice carrier ca provide advice on how to discharge patient without risking a claim o abandonment.

—doctors are often sued because the could not effect a cure. The publi tends to have greater expectations o medicine than are reasonable, and prudent doctor should clearly communi cate to the patient the limitations o prescribed treatment or surgery. Cau tious optimism is the watchword.

—obtaining consultations when indi cated not only helps the patient, but assists the doctor in avoiding being sued for failing to recognize and treat an unusual problem. Defense attorneys recommend that consultation is often advisable when a patient presents himself with a long-standing and unresolved ailment. And there is no harm in advising the patient that you want a consultation. If he declines, always annotate your chart.

—health care providers are being urged to report to their carrier any situation which may lead to a claim, including requests for medical records of patients who may have had a complicated course of treatment, or an expected post-op complication, or who have expressed dissatisfaction with the treatment they have received. Obtaining expert legal advice from defense counsel before a claim is made frequently helps to avoid a problem before it begins.

In every State, the definition of medical negligence is the failure to follow the standard of care exercised by other physicians in the same specialty, and practicing in the same locale. Every health care provider in Arizona is, therefore, obligated to keep himself apprised of what his colleagues are doing, of new directions in medicine, and of updated information about medications and surgery.

Finally, there is no substitute for care with caring. An honest, concerned, sympathetic attitude is your main prevention measure. Strive to develop it as that ounce of prevention that is worth much more than the pound of care.

J.F. Kahle, M.D., President ArMA

Mountain States Regional Hemophilia Center Program

Hemophilia is a hereditary blood disorder in which the deficiency of a factor in the blood prevents normal coagulation resulting in excessive internal and external bleeding. There are three types of hemophilia: Hemophilia A, or Classical Hemophilia, which is Factor VIII deficiency; Hemophilia B, or Christmas disease, Factor IX deficiency; and Hemophilia C, Factor XI deficiency. The first two are genetically transmitted as a sex-linked recessive trait, whereas Hemophilia C is transmitted as an autosomal recessive. Twenty percent to thirty percent of patients with hemophilia have no family history of the disorder. Hemophilia A is the most common of the three, B second most common, and Hemophilia C the least common. The hemophilas are further subdivided into mild, moderate, and severe, depending upon the clinical manifestations and the amount of coagulant activity present in plasma. In Arizona there are an estimated ninety patients with moderate or severe hemophilia.

The treatment of hemophilia has undergone marked changes over the last two decades. This has resulted largely from the increased availability of factor concentrates. It has been the impression of many that the clinical condition of the hemophiliac patient has improved over this time. However, in long-term studies of hemophiliacs, new complications have been recognized. These include chronic liver disease, immunological problems, hypertension, and renal disease. A recent survey has shown that in the United States spontaneous hemorrhage remains the leading cause of death, occurring in 45 percent of the patients. Of these, two-thirds are central nervous system bleeds with traumatic death (15 percent). Suicide (6 percent) is the next most frequent cause of death. Such studies question whether treatment programs are adequately educating patients to the need for early recognition and treatment of catastrophic bleeding, the need for careful followup, and the need for mental health care to prevent suicide. Although the median age of survival has increased over the last few years, hemophiliacs have a far from normal life span.

The current programs for the treatment of hemophilia can be subdivided into three categories. The first is demand infusion, that is, infusion given on an episodic basis at the time of each bleeding episode. This is generally preferred over prophylactic or maintenance infusion programs mainly because of the high cost of therapy and the fear that supplies of therapeutic materials may be overstrained. The second is home therapy. This method of health care delivery decreases cost, diminishes hospitalization time, decreases physician visits, and leads (in general) to a more normal life style. The third is comprehensive care, defined as the systematic application of various medical and paramedical disciplines. This approach to this chronic illness improves the health of the individual and conserves therapeutic resources.

The concept of a comprehensive care facility has developed because the treatment of hemophilia requires continuous monitoring, an awareness of all the community resources available to long-term care, and a multidisciplinary approach to help the patient cope with ordinary day-to-day activities. This year a total of 23 Comprehensive Centers have been identified in the United States. The University of Arizona Health Sciences Center has been awarded a federal grant to develop such a center for the State of Arizona. The concept of comprehensive care includes not only the evaluation and management of bleeding itself, but the management of the effects of bleeding and other aspects of hemophilia such as dental, orthopedic, physical and occupational therapy, mental health, and other services, all within the confines of a single institution.

The Mountain States Regional Hemophilia Center, based at the University of Arizona Health Sciences Center, will provide diagnostic treatment and consultative services for all health care providers treating patients with hemophilia. The Center will provide an environment for educational programs for professional and paraprofessional individuals who are caring for patients with hemophilia. The patient evaluation team will consist of: pediatric and internist hematologists, pediatric and adult orthopedic surgeons, dentists, physical therapists, social workers, blood bank pathologists, registered nurses, and a geneticist. The staff will work closely with existing programs in the State, such as the Treatment Center at St. Joseph's Hospital and Medical Center in Phoenix, Crippled Children's Services, etc. It is

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Marshall B. Block, M.D., Chairman
Fillmore K. Bagatell, M.D.
Walter V. Edwards, M.D.
Warren Gorman, M.D.
John R. Green, M.D.
John W. Kennedy, M.D.
George Lastnick, M.D.
William B. McGrath, M.D.
Stanley H. Nordomo, M.D.
Murray Spotnitz, M.D.
John Stoner III, M.D.
William C. Weese, M.D.
Donald J. Ziehm, M.D.

recommended that all hemophiliacs be evaluated in a comprehensive way at least once yearly. In addition to the medical, surgical, dental, and social evaluation of each patient, they will be checked yearly for the presence or absence of circulating anticoagulant, alterations in liver function, change in blood cell counts, and urine abnormalities. An ongoing record will be kept regarding the number of transfusions each patient requires per year, the amount of time lost from school or work, the financial burden the disorder places on the individual and/or family, and problems in acquiring optimal care for the condition.

Following the indepth comprehensive evaluation of the patient, the referring source will be provided with a concise summary of the Center's findings and a list of recommendations for improvement of the health care of the patient. The recommendations can be carried out in the referring source's own locale or, alternatively, the patient can be referred back to the Center. To register a patient in the Center or for additional information you may contact Dr. James J. Corrigan, Jr., Program Director, Department of Pediatrics, University of Arizona Health Sciences Center, Tucson, Arizona 85724, (telephone number: (602) 882-6527).

Louis Kettel, M.D., Dean

Auxiliary Highlights

Legislation *has* to be the most significant, threatening, controversial, unallurring helpful necessity ever to be simultaneously encouraged and ignored by doctors and their wives! This of course does not set the medical profession apart from most other entities in our peculiar society, for Legislation is commonly bypassed in the sweep of programs which quite legitimately demand attention. It may be the one element most capable of bringing out hostilities among otherwise reasonable friends, and perhaps that explains how intelligent people can risk their social "all" by just not getting involved in Legislative concerns.

The Arizona Auxiliary, however, continues to maintain a watch dog system of involvement, with designated leaders remaining vigilent and keeping the members informed. Such leaders are Ann Price and Ruth Ogden, who attended the A.M.A. sponsored Regional Conference on State Legislation and heard state departmental presentations of Health Services. Major concerns are V.D. Control, Nutrition for the Elderly, Immunization, and Comprehensive Health Planning.

State chairman Eloise Clymer gave dedicated attendance to the HEW hearing just as she sits countless meetings and hearings to qualify herself to spread the word among Auxiliary members. She works at motivating through communications with county chairmen, reporting at state board meetings, talking to legislators, and generally pushing for involvement in government via local and federal politics, helping with ARMPAC, and working to get out the vote. Selma Targovnik, M.D., chairperson of ArMA committee on Legislation, has welcomed Auxiliary chairmen to attend meetings which offer insight as to the legislative facts and opinions of the medical profession.

Involvement in politics would excuse no one, not even the most timid nor restriction conscious soul; there are many quietly effective means of involvement. Primarily, it is inexcusable to be uninformed, and numerous media offer condensed material for current up date. Legislative Roundup, Medical World News, A.M.A. News, and Arizona Medicine are valuable vehicles, and doctors are urged to take legislative materials home so that their wives can also be educated and informed as individuals.

Letters and personal contact with legislators are simple, effective ways to be involved with the mechanics of American government. Surely anyone can recognize the importance of participating, since we're each and all ingredients in the same stew.

Mrs. Melvin W. Phillips

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAV BEEN EDITED TO CONSERVE SPACE. A COMPLET COPY OF THE MINUTES OF ANY MEETING WILL B MAILED TO ANY MEMBER REQUESTING THEM.

MALPRACTICE INSURANCE CRISIS COMMITTEE

Meeting of the Malpractice Insuranc Crisis Committee of the Arizona Medic Association, Inc., held Thursday, Octobe 13, 1977, at 810 West Bethany Home Roac Phoenix, Arizona convened at 7:41 p.m Vernor F. Lovett, M.D., Chairman, pre siding.

INTRODUCTION

Dr. Lovett welcomed the two new mem bers of the Committee present, Drs. Mage and Winograd.

REVIEW OF H.B. 2001

Dr. Lovett, Mr. Jacobson and Mr. Neilse reviewed and answered questions pertainin to the passage of H.B. 2001 on February 27 1976 and the constitutional challenges whic have been successfully defended with th exception of the posting of bond. RE CEIVED FOR INFORMATION.

STUDY COUNCIL ON TORTS & LIABILITY INSURANCE

Dr. Lovett reported on the activities of th Study Council on Torts & Liability Insur ance which has expanded its role during th last legislative session. He explained tha the 3rd session of the Council is to meet o Friday, October 14th with representatio from the Insurance Industry and acturaria presentation by Woody Beckman on th agenda. The next session will take place o Friday, November 18, during which tim Judge Broomfield and members of the Ba are to make their presentations. RECEIVEI FOR INFORMATION.

COUNTERSUITS

Dr. Lovett explained that the Ad Ho Malpractice Insurance Crisis Committee ha considered, during a previous meetin whether the Association should becom actively involved in assisting countersuit that might be brought in response t unfounded malpractice litigation. After re ceipt of letter from counsel, Mr. John J Bauma, firm of Snell & Wilmer, in which h reported in part "to the extent that th Arizona Medical Association, Inc. woul actually encourage or support countersuit that are not well founded, the Associatio itself could find itself as a defendant expose to damages". The Board of Trustees of th Mutual Insurance Company of Arizon considered their role in countersuits. Dr Brooks informed the Committee that th MICA Board sought legal advise an determined that there was currently no rol for the insurance company to play in th arena of countersuits. Considerable discus sion ensued as to the value of countersuits a an education tool as well as being in position to inform and advise members wh feel that they have been involved in a unfounded malpractice litigation.

IT WAS MOVED AND CARRIE THAT THE COMMITTEE REVIEW EX PERIENCES OF OTHER STATE AS

SOCIATIONS INVOLVED IN A COUNTERSUIT PROGRAM, REQUEST DATA FROM THE AMERICAN MEDICAL ASSOCIATION AND TO DETERMINE THE ASSOCIATION'S LEGAL JEOPARDY SHOULD WE ENCOURAGE OR SUPPORT COUNTERSUITS.

CONSULTATION FOR THE PLAINTIFF

Dr. Lovett reported that many physicians appear to be unaware of the requirement outlined in H.B. 2001 under 12-567 pertaining to medical liability review panels that "B 6) The superior court shall provide to any party upon request the lists of licensed health care providers maintained by such clerk for the purposes of this section. A party may then select any one of the specialists on such lists and such specialists shall discuss the case with the claimant and his attorney." The plaintiff attorneys are still maintaining that there is a conspiracy of silence as suggested by Mr. Robert Meyers, Plaintiff Attorney, during his presentation to the Study Council on Torts & Liability Insurance September 16, 1977. Further, Dr. Lovett reported that there appears to be a shopping by the plaintiff attorneys of consultants until they find one that may agree with their case, and compensation of consultants was not addressed in the legislation. Discussion ensued as to the importance of publicizing the requirements outlined in H.B. 2001, establishing proper criteria for serving as a consultant through the list provided by the Board of Medical Examiners as well as obtaining the opinion of the court in this matter.

IT WAS MOVED AND CARRIED THAT THE COMMITTEE SEEK THE OPINION OF JUDGE BROOMFIELD, PRESIDING JUDGE AT THE MARICOPA COUNTY SUPERIOR COURT CONCERNING THE LISTING OF PHYSICIANS PROVIDED BY BOMEX FOR THE PURPOSE OF PANEL MEMBERS AS WELL AS CONSULTANTS, DETERMINE CRITERIA FOR SERVING ON SUCH A LIST AND THEN PUBLICIZE THE REQUIREMENT AS OUTLINED IN 12-567.B(6).

CONSTITUTIONAL AMENDMENTS

Dr. Lovett presented the following thoughts for stabilizing the liability insurance which may necessitate amendments to the State constitution:

1. Limit pain and suffering to an amount certain i.e., $25,000.
2. Limit total awards to an amount certain i.e., $150,000 to $500,000.
3. No fault insurance.
4. Arbitration—binding.
5. Eliminate punitive damages.
6. Itemized verdicts.
7. Periodic payments.

During discussion of the recommendations as outlined above, it was pointed out that any limitation on awards or suggestions which we may find will need an amendment to the State constitution should not be just on medical malpractice but be on all liability awards.

IT WAS MOVED AND CARRIED TO RETAIN MR. WOODY BECKMAN, ACTUARY, TO DETERMINE THE EFFECTS ON LIMITING PAIN AND SUFFERING AWARDS TO $25,000 OR ANOTHER AMOUNT CERTAIN AND/OR LIMIT TOTAL AWARDS TO AN AMOUNT CERTAIN I.E., $150,000 TO $500,000, A NO FAULT INSURANCE PROGRAM AS BEING DISCUSSED IN CALIFORNIA OR ANY OTHER AREA OF ASSISTANCE THAT MR. BECKMAN MAY CONSIDER OF VALUE.

RESEARCH GRANT

Dr. Lovett explained to the Committee that Marsh & McLennan have provided a research grant of $5,000 per quarter to the Association for investigating causes of malpractice and that the total received to date is $25,000. Discussion ensued as to how best to utilize the grant from Marsh & McLennan.

IT WAS MOVED AND CARRIED THAT A SUB COMMITTEE WHICH INCLUDES DRS. LOVETT AND BROOKS BE DISPATCHED TO DISCUSS FUNDING A STUDY OF MEDICAL MALPRACTICE UNDER THE AUSPICIES OF THE SUPREME COURT OF ARIZONA WITH CHIEF JUSTICE DUKE CAMERON.

GENERIC SCREENING

Dr. Lovett explained that the Mutual Insurance Company of Arizona is undertaking a pilot generic screening process as has been developed for California Medical Association. A paper on the generic screening process was distributed to the committee and is on file in the Association's headquarters office for information.

SCIENTIFIC ASSEMBLY COMMITTEE

The meeting of the Scientific Assembly Committee of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona on Saturday, October 15, 1977, convened at 1:21 p.m., Richard A. Silver, M.D., Chairman, presiding.

CALIFORNIA COLLEGE OF MEDICINE FACULTY

Extensive review was made of the letter from Robert C. Combs, M.D., Associate Dean, California College of Medicine, University of California, Irvine, dated September 29, 1977 in which he set forth his proposal for our 1978 meeting.

Because of space limitations, it was determined to not include Drs. Mull, Tobis, and Wilbert. Many variations and possibilities were discussed and several changes were made in the proposal culminating in the following proposal:

	Tucson Com'y Ctr	*Tucson Com'y Ctr*	*Tucson Com'y Ctr*	*Tucson Com'y Ctr*
Thursday A.M.	**Apache Rm [75]**	**Cochise Rm [75]**	**Maricopa Rm [225]**	**Mohave Rm [225]**
8-9:00 A.M.				"Dermatology"
9-9:30 A.M.	"Diabetes and Hypoglycemia"		'Nephrology"	
	Grant Gwinup, M.D.		Leonard B. Beruman, M.D.	Orville J. Stone, M.D.
9:30-10 A.M.				*A six-hour post-graduate course. Advance registration required. Starts at 8 AM & concludes at 5 PM*
10-10:30 A.M.				
	Moderator:		Moderator:	
10:30-11 A.M.	Recess - Exhibits	Recess - Exhibits	Recess - Exhibits	Recess - Exhibits
11-11:30 A.M.		"Treatment of Leukemia-Acute and Chronic"	"Nephrology"	"Dermatology"
		Lewis M. Slater, M.D.	Leonard B. Berman, M.D.	Orville J. Stone, M.D.
				A six-hour post-graduate course. Advance registration required. Starts at 8 AM & concludes at 5 PM

11:30-12 Noon				
		Moderator:	Moderator:	
12 Noon-2 P.M.	Lunch	Lunch	Lunch	Lunch
Thursday P.M.	*Tucson Com'y Ctr* **Apache rm [75]**	*Tucson Com'y Ctr* **Cochise Rm [75]**	*Tucson Com'y Ctr* **Maricopa Rm [225]**	*Tucson Com'y Ctr* **Mohave Rm [225]**
2-2:30 P.M.	"Hypoxemia As Seen Through Arterial Blood Gases"	'Neonatal Infections"	"Radiology of the G.U. Tract"	"Dermatology"
	Archie F. Wilson, M.D.	Gerald R. Greene, M.D.	Leonard B. Berman, M.D. Peter Dure-Smith, M.D. ?sp	Orville J. Stone, M.D.
2:30-3 P.M.				*A six-hour post-graduate course. Advance registration required. Starts at 8 AM & concludes at 5 PM*
	Moderator:	Moderator:	Moderator:	
3-3:30 P.M.	Recess - Exhibits	Recess - Exhibits	Recess - Exhibits	Recess - Exhibits
3:30-4 P.M.	"Pathophysiology Of Asthma"	"Susceptibility to Malignant Diseases:-Genetics, -Chemical Markkers, -Radiation, -Drug Induction"	"Nephrology"	"Dermatology"
	Archie F. Wilson, M.D.	Lewis M. Slater, M.D.	Leonard B. Berman, M.D.	Orville J. Stone, M.D.
	Moderator			*A six-hour post-graduate course. Advance registration required. Starts at 8 AM & concludes at 5 PM*
4-4:30 P.M.	"Treatment Of Asthma"			
	Archie F. Wilson, M.D.			
	Moderator:			
4:30-5 P.M.	"Pulmonary Re-Habilitation-What Can Be Done"			
	Archie F. Wilson, M.D.			
	Moderator:	Moderator:	Moderator:	
Friday A.M.	*Tucson Com'y Ctr* **Apache Rm [75]**	*Tucson Com'y Ctr* **Cochise Rm [75]**	*Tucson Com'y Ctr* **Maricopa Rm [225]**	*Tucson Com'y Ctr* **Mohave Rm [225]**
8-9:00 A.M.				"Gastroenterology"
9-9:30 A.M.	"Radiology Of Pregnancy And Ovarian Vein Syndrome"			"Cardi
	Peter Dure-Smith, M.D. ?sp			
9:30-10 A.M.				
10-10:30 A.M.				
	Moderator:			Modera
10:30-11 A.M.	Recess - Exhibits			Recess
11-11:30 A.M.	"Thyroid"			"Cardi
	Grant Gwinup, M.D.			
11:30-12 Noon				
	Moderator:			Modera
12 Noon-2 P.M.	Lunch			Lunch
Friday P.M.	*Tucson Com'y Ctr* **Apache Rm [75]**			*Tucson* **Cochise**
2-2:30 P.M.	"Obesity"			"Cardi
	Grant Gwinup, M.D.			
2:30-3 P.M.				
	Moderator:			Modera
3-3:30 P.M.	Recess - Exhibits			Recess

	Sheldon Zinberg, M.D.		
	A six-hour post-graduate course. Advance registration required. Starts at 8 AM & concludes at 5 PM		
ıibits	Recess - Exhibits	Recess - Exhibits	
	"Gastroenter-ology"		
	Sheldon Zinberg, M.D.		
	A six-hour post-graduate course. Advance registration required. Starts at 8 AM & concludes at 5 PM		
	Lunch	Lunch	
Ctr	*Tucson Com'y Ctr*	*Tucson Com'y Ctr*	
]	**Maricopa Rm [225]**	**Mohave Rm [225]**	
	"Psychiatry For The Non-Psychia-trist Physician"	"Gastroenter-ology"	
	Donald W. Schafer, M.D.	Sheldon Zinberg, M.D.	
	A six-hour post-graduate course. Advance registration required. Starts at 8 AM & concludes at 5 PM		
	Moderator:		
its	Recess - Exhibits	Recess - Exhibits	

3:30-4 P.M.	"Intestinal Parasites In Children" title to be changed	"Psychiatry For The Non-Psychiatrist Physician"	"Gastroenterology And Radiology Of The G.I. Tract"
	Gerald R. Greene, M.D.	Donald W. Schafer, M.D.	Sheldon Zinberg, M.D. Edward R. Dana, M.D.
			A six-hour post-graduate course. Advance registration required. Starts at 8 AM & condludes at 5 pm
4-4:30 P.M.			
		Moderator:	
4:30-5 P.M.	"Identifying The Causative Organism Before The Culture Returns"		
	Gerald R. Greene, M.D.		
	Moderator:	Moderator:	

COMPUTER ASSISTED INSTRUCTION

It was moved and carried to make the subject item a special topic as part of the 1978 program which will be located in the hotel from 9 a.m. to 12 noon and 2 p.m. to 5 p.m. on Thursday and Friday, April 27 and 28, 1978 under the direction of Dr. Combs.

LOCAL PAPERS

Several suggestions from local speakers were offered to the committee which decided to delay action until the next meeting with the exception of the proposal to provide a small day-long workshop on hypnosis which was turned down due to lack of space.

BREAKFAST PANELS

Thursday - April 27th

IT WAS MOVED AND CARRIED TO SCHEDULE A PANEL ON "THE DISTRESSED PHYSICIAN: WHERE DO WE DO FROM HERE" AT THIS TIME.

Friday - April 28th

IT WAS MOVED AND CARRIED TO ASK DR. SCHAFER OF THE CALIFORNIA COLLEGE OF MEDICINE TO PROVIDE A PROGRAM THAT WOULD BE OF INTEREST TO BOTH THE PHYSICIAN AND HIS SPOUSE.

GOLF-TENNIS TOURNAMENTS

IT WAS MOVED AND CARRIED THAT STUART I. HOLTZMAN, M.D., BE ASKED TO CHAIR THE GOLF TOURNAMENT AND THAT SAMUEL J. GRABB, M.D., BE ASKED TO CHAIR THE TENNIS TOURNAMENT.

FUTURE MEETINGS

Mr. Robinson reported that the Board of Directors has approved the new format as proposed by this committee to start with the 1979 meeting. Also, the Board approved the Scottsdale Registry for the 1980 meeting and reported that they had eliminated the registration fee for those attending only the House of Delegates meetings.

The committee also advised that the University of Arizona College of Medicine had expressed an interest in providing the faculty for the 1980 meeting.

COMMERCIAL RECORDINGS

It was agreed that we would not seek to have the scientific sessions recorded for the purpose of selling the tapes.

MODERATORS

It was suggested that we ask the University of Arizona College of Medicine if they would be interested in providing moderators for our 1978 meeting.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona on Friday, October 21, 1977 convened at 6:41 p.m., John F. Kahle, M.D., President and Chairman, presiding.

AD HOC COMMITTEE ON HEALTH CARE COSTS

Dr. Hastings reported in some depth on the deliberations of his committee indicating that they met twice and are planning a joint meeting with the Arizona Hospital Association's committee. Many aspects of the problem have been discussed with only one firm proposal settled upon to date. The recommendation is as follows:

"IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE ASSOCIATION REQUEST THE HOSPITALS TO PROVIDE A SUMMARY OF CHARGES ON EACH PATIENT UPON DISCHARGE AND AFFIX IT TO THE HOSPITAL CHART FOR REVIEW BY THE ATTENDING PHYSICIAN. FURTHER, THAT THE BOARD OF DIRECTORS CONSIDER INTRODUCING A RESOLUTION TO THE HOUSE OF DELEGATES IN SUPPORT OF THIS MATTER DURING THE NEXT ANNUAL MEETING."

COUNCIL OF PROFESSIONS

Dr. Chisholm reviewed the activities and accomplishments of the Council of Professions since its inception indicating its importance to our legislative contacts and legislative program.

IT WAS MOVED AND CARRIED TO REAFFIRM THE ASSOCIATION'S SUPPORT OF THE ARIZONA COUNCIL OF THE PROFESSIONS, INC.

SUGGESTED GUIDELINES FOR HOSPITALS' BILLING OF INDEPENDENT PHYSICIANS

Mr. Robinson reported on the current status of the subject problem stating that there appears to be a solution developing among the various attorneys involved.

MICC

Mr. Barnett reported on the current activities of the Malpractice Insurance Crisis Committee indicating that they are currently investigating possible countersuit activity, problems of malpractice panel members, providing consultants for plaintiffs' attorneys, and a possible research grant to assist the Supreme Court of Arizona in conducting research into the malpractice panel system.

ARIZONA HEALTH SERVICE CORPS

Financial

Mr. Robinson reported that, to date, $82,229.41 had been expended on the AHSC proposal. Another $16,293.79 had been committed but unexpended for a total of $98,523.20 committed and spent out of the $101,349 contract leaving an unexpended balance of $2,825.80. He pointed out that the average monthly expenses run $5,779.71 which means that we would not have funds to pay for a month's extension, even though one was granted.

Relocation Expenses

The subject of compensating Ward B. Studt, M.D., for relocation expenses was reviewed. Dr. Studt stated he did not expect to be reimbursed for his relocation expenses unless the contract was extended to the second year.

Proposal Rejection

Dr. Studt reported on a telephone conference with Fitzhugh Mullan, M.D., and Mr. Howard Hilton of the National Health Service Corps at HEW in Washington, D.C., which occurred about 1 p.m. this date.

During this call, Dr. Studt was advised that our proposal as presented on July 26, 1977 had been rejected and would not be funded. He stated that there might possibly be funds in the neighborhood of $100,000 to be used in the following three ways: (1) providing a recruiting service for NHSC sites, (2) providing a continuing education service to existing NHSC sites, and (3) providing funds for residency activity in existing or new NHSC sites.

Considerable discussion ensued on the various ramifications of this alternate proposal, culminating in the following three actions:

IT WAS MOVED AND CARRIED TO ACCEPT THE VERBAL REJECTION OF THE FOUNDATION'S PROPOSAL (AS SUBMITTED TO MRS. GYNETHA SHACKELFORD, PROJECT OFFICER, ON JULY 26, 1977) FOR THE SECOND YEAR FUNDING OF THE ARIZONA HEALTH SERVICE CORPS AS VERBALLY RELATED TO WARD B. STUDT, M.D., BY FITZHUGH MULLAN, M.D., DIRECTOR, NATIONAL HEALTH SERVICE CORPS.

IT WAS MOVED AND CARRIED TO NOT ASK FOR AN EXTENSION OF THE CONTRACT PAST OCTOBER 31, 1977.

IT WAS MOVED AND CARRIED TO SUPPORT THE UNIVERSITY OF ARIZONA SHOULD THEY WISH TO PURSUE THE ALTERNATE PROPOSAL OF (1) PROVIDING A RECRUITING SERVICE FOR NHSC SITES, (2) PROVIDING A CONTINUING EDUCATION SERVICE TO EXISTING NHSC SITES, AND (3) PROVIDING FUNDS FOR RESIDENCY ACTIVITY IN EXISTING OR NEW NHSC SITES.

STUDENT FUNDING

IT WAS MOVED AND CARRIED TO AUTHORIZE $80 TO ASSIST IN SENDING A STUDENT TO THE DENVER MEETING OF THE AMERICAN MEDICAL WOMANS ASSOCIATION.

MILEAGE REIMBURSEMENT

Mr. Robinson reported on his research regarding updating the amount we reimburse people for the use of their cars on Association business.

IT WAS MOVED AND CARRIED TO ESTABLISH 20 CENTS AS THE PER MILE REIMBURSEMENT RATE EFFECTIVE OCTOBER 22, 1977.

PSRO

Neil O. Ward, M.D., reported that the Maricopa County Medical Society had accepted a planning grant to develop a Professional Standards Review Organization for the northern half of the state.

SPECIAL LEGISLATIVE COMMITTEE

The meeting of a special Committee to Discuss Optometrists' Attempts to Practice Medicine of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona on October 26, 1977 convened at 6:52 p.m., John F. Kahle M.D., President and Chairman, presiding.

PURPOSE

Dr. Kahle introduced all participants and stated that the purpose of the meeting was to discuss strategy regarding possible proposed state legislation that would permit optometrists to use drugs.

BACKGROUND

Dr. Moore reviewed for the committee his very detailed 13-page chronology of events during the 1977 legislative session relating to House Bill 2151 and Senate Bills 1055 and 1062.

PLANS

General discussion ensued with several suggestions being made: (1) entering into a program where all physicians would attempt to get their patients to write their legislators asking them to oppose legislation that would give optometrists the right to use drugs; (2) letters to ArMA members to educate them to the problem posed by this attempt to legislate the right to practice medicine to non-physicians.

PROCEDURE AND PROTOCOL

It was pointed out that the Arizona Ophthalmological Society had engaged Mr. Charles L. Strouss, Jr. to be the lobbyist for this particular matter.

It was agreed that Mr. Strouss would be responsible for directing and coordinating all aspects of the lobbying effort during the coming legislative session and that he, Mr. Strouss, would call upon the Arizona Medical Association for assistance as he felt the need to do so.

All agreed that this concept of having one person direct the lobbying effort was most essential to eliminate possible confusion, duplication, and misunderstanding.

Mr. Robinson reported that Mr. Strouss had, during a conversation on September 15, 1977, agreed to be the leader in this effort and was agreeable to this general approach. It was understood that the Association would not take the initiative in any activity regarding this entire effort, but would respond as best it could when so requested by the AOS and Mr. Strouss.

PHYSICIAN REHABILITATION COMMITTEE

The meeting of the Physician Rehabilitation Committee of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona on Saturday, November 5, 1977, convened at 12:12 p.m., Thomas E. Bittker, M.D., Chairman, presiding.

CONTACT TRAINING

It was determined that the next meeting would be devoted primarily to training on how to handle contacts with physicians who are having problems. The video tape on this matter is to be sought from the Washington State Medical Association. The next meeting will be held Saturday, January 28, 1978.

IMMUNITY STATUTES

Mr. Robinson pointed out that the Association was successful in getting immunity legislation passed that directly effect this committee. The following is an excerpt from the Association's legal opinion of this statute.

"A.R.S. Sections 36-2351, 2352 are directly applicable providing immunity not only for members, agents or employees of health

care peer review committees, but also for those who furnish records, information or assistance to such committees. The statutes are quite clear in this regard and it might be well to set them forth herein.

A.R.S. Section 36-2351.1(a)(b) reads as follows:

"36-2351. *Definitions*

In this Article, unless the contest otherwise requires:

1. "Health Care Peer Review Committee" means any organizational structure of a professional society of licensed health care providers, the purpose of which is one of the following:
 (a) To evaluate and improve the quality of health care.
 (b) To review and investigate the conduct of licensed health care providers in order to determine whether disciplinary sanctions should be imposed."

A.R.S. Section 36-2352 reads as follows:

"36-2352. *Health care peer review committees; immunity*

A person who, in good faith and without malice, takes any action or makes any decision or recommendation as a member, agent or employee of a health care peer review committee or who furnishes any records, information or assistance to such a committee shall not be subject to liability for civil damages or any legal action in consequence of such action, nor shall the professional society which established such committee or their officers, directors, employees or agents of such professional society be liable for the activities of any such person. This section shall not be construed to relieve any person of liability from treatment of a patient."

MICA DEFENSE

The question was raised as to whether the Mutual Insurance Company of Arizona would defend a physician who reports to this committee and is sued for so reporting. The company (MICA), in a letter dated October 25, 1977, said, in part, the following.

". . . it would be more than adequate to advise any physician who inquires that the MICA policy would provide a defense to an insured MICA physician who reports another physician for incompetency, etc. to a formal committee of the medical society or hospital."

MEETINGS

Dr. Bittker reported on informational meetings that have been held with the medical staffs of several hospitals and with the president of the Maricopa County Medical Society. Dr. Scheiber reported on his plans to talk with students and housestaff in the Tucson area. It was determined to contact the Directors of Medical Education to advise them of the existence and purpose of the committee.

CASE REVIEWS

A review of seven outstanding cases was made closing one of them with continued follow up on the remaining six.

Meeting adjourned 2:46 p.m.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona, on Friday, November 18, 1977 convened at 6:47 p.m., John F. Kahle, M.D., President and Chairman, presiding.

MINUTES

ADVANCED CARDIAC LIFE SUPPORT

Frederick Scott, M.D., made a presentation to the Executive Committee concerning his activities in advanced cardiac life support. He informed the committee that there are currently five physicians in Arizona certified by the American Heart Association as instructors for advanced courses in C.P.R. It is Dr. Scott's intention to provide programs not only to emergency room nurses and paramedics in Arizona, but to instruct physicians in advanced cardiac life support as well as instruct a cadre of physicians who could be certified as instructors. Dr. Scott seeks permission to utilize the Association headquarters office for some of the programs as well as Association support of the activity. Further, he informed the Executive Committee that equipment to put on the program will cost in the neighborhood of $13,000.

IT WAS MOVED AND CARRIED TO REFER DR. SCOTT'S ADVANCED CARDIAC LIFE SUPPORT PROGRAM TO THE PROFESSIONAL COMMITTEE FOR REVIEW AND EXPLORATION OF SOME METHOD OF FUNDING WITH REPORT TO THE BOARD OF DIRECTORS DURING ITS DECEMBER 17TH MEETING.

ARIZONA HEALTH SERVICE CORPS

Ward Studt, M.D., reported to the Committee that efforts to continue the Arizona Health Service Corps under the auspices of the University of Arizona, as suggested during the last Executive Committee meeting, was not possible. Therefore, the offices of the Arizona Health Service Corps were terminated effective October 31, 1977. Dr. Studt extended his appreciation to the Executive Committee and the Arizona Medical Association for their part in the development of the proposed Arizona Health Service Corps. RECEIVED FOR INFORMATION.

ARIZONA COUNCIL OF PROFESSIONS

Dr. Chisholm informed the Executive Committee that during the Council of Professions meeting held on Wednesday, November 16, the State Bar represented by Walter Cheifetz made presentation concerning their current activities in professional liability and need for further tort reforms. Although the State Bar's Committee on Professional Liability had not yet developed any specifics, they were asking the Council of Professions to indicate an interest in working with them in seeking appropriate tort reforms. Dr. Manley also attended the Council of Professions meeting and supports the State Bar's suggestion that tort reforms be introduced of interest to all the professions.

IT WAS MOVED AND CARRIED TO INFORM THE STATE BAR OF ARIZONA THAT THE ASSOCIATION WOULD SUPPORT SEEKING A COMMON GROUND AND INDICATE OUR INTEREST IN PURSUING A COOPERATIVE VENTURE.

RATE REVIEW FOR HOSPITAL BASED PHYSICIANS

By letters of November 10 and November 16, 1977, Mr. Jacobson informed the Executive Committee of continued discussion with the Department of Health Services and The Arizona Hospital Association concerning rate review for hospital based physicians. Mr. Jacobson's letters were received for information with the suggestion that Mr. Jacobson update the Executive Committee during its next session.

MERIT SYSTEM SELECTION FOR NEW APPOINTMENTS TO BOMEX PROTECTION FOR PEER REVIEW

Maricopa County Medical Society's Board of Directors respectfully request that ArMA consider introducing legislation addressing the following issues:

1. *Merit system selection for new appointments to BOMEX.*
 Current Arizona Statute states "the governor shall appoint "members to the Board of Medical Examiners. This affords the opportunity for a respected panel of professionals to become political cronies ultimately. A Committee composed of ArMA members could be mandated by law to prepare a list of nominees for BOMEX by mechanisms similar to the present method of nominating appellate court judges.
2. *Protection for peer reviewers.*
 Although MICA pays defendant physician costs, there is no deterrent to filing suit for disgruntled physicians who have been disciplined by their colleagues. If plaintiffs were required to pay defense costs of defendant Committee members, frivolous suits filed only to assuage injured egos might be less likely.

It was determined to present the above mentioned request by the Maricopa County Medical Society to legal counsel for review and drafting of legislation for presentation to the Legislative Committee for their consideration.

REVIEW OF AMA INTERIM MEETING ACTIVITIES

The Executive Committee reviewed with the Delegates to AMA the reports and resolutions introduced thus far for consideration during the interim meeting to be held December 4 through December 7, 1977 in Chicago. In that the Delegates Handbook had been received on this date by most of the Delegates, it was not possible to go in to a great amount of detail, except for review.

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

THE FOLLOWING INSTITUTIONS AND ORGANIZATIONS HAVE RECEIVED ACCREDITATION FOR CONTINUING MEDICAL EDUCATION FROM THE COUNCIL ON MEDICAL EDUCATION OF THE AMA

ARIZONA STATE HOSPITAL PHOENIX
DESERT SAMARITAN HOSPITAL MESA
GOOD SAMARITAN HOSPITAL PHOENIX
HEALTH MAINTENANCE ASSOCIATES
PHOENIX INDIAN MEDICAL CENTER
MARICOPA COUNTY GENERAL HOSPITAL PHOENIX
MEMORIAL HOSPITAL PHOENIX
ST. LUKE'S HOSPITAL AND MEDICAL CENTER PHOENIX
ST. JOSEPH'S HOSPITAL AND MEDICAL CENTER PHOENIX
TUCSON HOSPITALS MEDICAL EDUCATION PROGRAM TUCSON
U. OF A. HEALTH SCIENCES CENTER
VETERANS ADMINISTRATION CENTER PRESCOTT
VETERANS ADMINISTRATION HOSPITAL PHOENIX

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category I credit. Physicians who participate in programs which are designated Category I by accredited institutions will receive Category I credit toward the ArMA Certificate in CME and the AMA's Physicians Recognition Award.

FEBRUARY

COMBINED GRAND ROUNDS
February 4, 1978, Scottsdale Memorial Hospital. Sponsor: Family Practice Residency Programs of St. Joseph's, Good Samaritan, and Scottsdale Memorial Hospitals. Contact: Ann Larsen, R.N., Program Coordinator, Family Practice Residency Program, St. Joseph's Hospital and Medical Center, Phoenix, AZ 85013. Approved for required hours toward the AMA Certificate in Continuing Medical Education.

SOUTH AMERICAN MEDICAL SEMINAR
January 29 to February 7, 1978. Lima, Peru & Quito, Equador. Sponsor: International Seminars. Contact: Wei-Ping Loh, M.D., Ph.D., 120 S. Central Ave., St. Louis, Missouri 63105. Approved for hour elective hours toward the ArMA Certificate in Continuing Medical Education.

AAFP ANNUAL MEETING 1978
February 1-4, 1978. Doubletree Inn, Scottsdale, AZ. Sponsor: Arizona Academy of Family Physicians. Contact: Mrs. June Boykin, 1027½ E. Camelback Rd., Phoenix, AZ 85014. Approved for required hours toward the ArMA Certificate In Continuing Medical Education.

2ND INTERNATIONAL CONFERENCE ON INTEGRATED CANCER MANAGEMENT
February 22-25, 1978. Carefree Inn & Resort, Carefree, AZ. Sponsor: Good Samaritan Hospital, Div. of Oncology. Contact: Medical Staff Service Office, GSH, P.O. Box 2989, Phoenix, AZ 85062. Approved for 16 required hours toward ArMA CME Certificate.

GYNECOLOGIC ONCOLOGY AND SLIDE SEMINAR
February 4-5, 1978. Marriott Hotel, Tucson, AZ. Sponsor: Arizona Society of Pathologists. Contact: Executive Secretary, Arizona Society of Pathologists, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 7 required hours toward the ArMA Certificate in Continuing Medical Education.

SOUTHWESTERN SEMINAR ON NEUROMUSCULAR DISORDERS IN CHILDREN
February 17-18, 1978. Health Sciences Center, Main Auditorium, Tucson, AZ. Sponsor: U of A Health Sciences Center Dept. of Neurology. Contact: Lawrence Z. Stern, M.D., Dept. of Neurology, U of A Health Sciences Center, Tucson, AS 85724. Approved for 9 required hours toward the ArMA Certificate in Continuing Medical Education.

ARIZONA SOCIETY OF ANESTHESIOLOGISTS ANNUAL MEETING
February 25 & 26, 1978. Scottsdale Hilton Hotel, Scottsdale, AZ. Sponsor: Arizona Society of Anesthesiologists. Contact: Casey D. Blitt, M.D., Dept. of Anesthesiology, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 9 required hours toward the ArMA Certificate in Continuing Medical Education.

MARCH

VALLEY OF THE SUN DERMATOLOGIC SYMPOSIUM
March 2-5, 1978. Casa Blanca Inn, Scottsdale, AZ. Sponsor: Phoenix Dermatological Society, & University of Arizona. Contact: Dr. Fillmore K. Bagatela, Dr. Peter J. Lynch, 333 E. Virginia, Phoenix, AZ. Approved for 12 required hours toward the ArMA Certificate in Continuing Medical Education.

DAY OF HEMATOLOGY
March 3, 1978, Camelback Inn, Scottsdale, AZ. Sponsor St. Joseph's Hospital and Medical Center, Phoenix, AZ. Contact. Ethelann Murray, M.D., St. Joseph's Hospital and Medical Center, 350 West Thomas Road, Phoenix, AZ 85013. Approved for 6 required hours toward the ArMA Certificate in Continuing Medical Education.

THE SECOND NATIONAL CONFERENCE ON OTITIS MEDIA
March 3-5, 1978, Doubletree Inn, Scottsdale, AZ. Sponsor Univ. of Arizona College of Medicine and the Indian Health Service Contact Office of Continuing Medical Education and Outreach, University of Arizona College of Medicine, Tucson, AZ 85724. Approved for 17.5 required hours toward the ArMA Certificate in Continuing Medical Education.

NEW MEXICO METABOLISM SYMPOSIUM
March 9, 10 & 11, 1978. Taos, New Mexico. Sponsor: Taos, New Mexico Sponsor: Taos Metabolism Symposium Contact: Office of Education, Lovelace Bataan Medical Center, Albuquerque, New Mexico.

PURGATORY COLORADO MEDICAL SK SYMPOSIUM. "GERIATRICS: Diagnosi and Treatment".
March 10-11, 1978. Durango, Co. Sponsor New Mexico Academy of Family Physicians. Contact: Albuquerque Academy of Family Physicians, 2650 Yale, S.E., Albuquerque, N.M. 87106. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

2ND ANNUAL SEMINAR, CURRENT CONCEPTS OF HYPERTENSION.
March 11-12, 1978. Scottsdale Registry Resort, Scottsdale, AZ. Sponsor: Health Maintenance Associates. Contact: Dr. L. A. Oslen, Health Maintenance Associates 4747 N. 22nd St., Phoenix, AZ 85016 Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

CURRENT CONCEPTS IN CEREBROVASCULAR DISEASE.
March 11-18, 1978. Sun Valley Inn, Sun Valley, Idaho. Contact: Margo Walter Administrative Assistant, Office of Continuing Medical Education and Outreach U of A Health Sciences Center, Tucson AZ 85724. Approved for 22.5 required hours toward the ArMA Certificate in Continuing Medical Education.

GASTROENTEROLOGY FOR CLINICIAN — 1978
March 13-15, 1978. Del Webb's Towne House, Phoenix, AZ. Sponsor: American College of Gastroenterology & the Institute of Gastroenterology, Good Samaritan Hospital. Contact: David C. H. Sun, M.D Good Samaritan Hospital, Phoenix, AZ 85036. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

"MANDATORY REPORTING OF CHILD ABUSE"
March 18, 1978, Arizona Medical Association offices, Phoenix. Sponsor: Psychiatric Services, Ltd. Contact: Robert T. Dean, Jr., M.D., Ste. 121, 2202 E. Camelback Rd., Phoenix, AZ 85016.



Vertigo spoils the view.

■ **Most Widely Prescribed**—Antivert is the most widely prescribed agent for the management of vertigo* associated with diseases affecting the vestibular system such as Menière's disease, labyrinthitis, and vestibular neuronitis.

■ **Relief of Nausea and Vomiting**—Antivert/25 can relieve the nausea and vomiting often associated with vertigo.*

■ **Dosage for Vertigo*** —The usual adult dosage for Antivert/25 is one tablet t.i.d.

BRIEF SUMMARY OF PRESCRIBING INFORMATION

INDICATIONS. Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: Management of nausea and vomiting and dizziness associated with motion sickness.

Possibly Effective: Management of vertigo associated with diseases affecting the vestibular system.

Final classification of the less than effective indications requires further investigation.

CONTRAINDICATIONS. Administration of Antivert (meclizine HCl) during pregnancy or to women who may become pregnant is contraindicated in view of the teratogenic effect of the drug in rats.

The administration of meclizine to pregnant rats during the 12-15 day of gestation has produced cleft palate in the offspring. Limited studies using doses of over 100 mg./kg./day in rabbits and 10 mg./kg./day in pigs and monkeys did not show cleft palate. Congeners of meclizine have caused cleft palate in species other than the rat.

Meclizine HCl is contraindicated in individuals who have shown a previous hypersensitivity to it.

WARNINGS. Since drowsiness may, on occasion, occur with use of this drug, patients should be warned of this possibility and cautioned against driving a car or operating dangerous machinery.

Usage in Children. Clinical studies establishing safety and effectiveness in children have not been done; therefore, usage is not recommended in the pediatric age group.

Usage in Pregnancy. See "Contraindications."

ADVERSE REACTIONS. Drowsiness, dry mouth and, on rare occasions, blurred vision have been reported.

More detailed professional information available on request.

ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017

Antivert®/25
(meclizine HCl) 25 mg. Tablets
for vertigo*



THE STRENGTH TO OVERCOME EXCESSIVE ANXIETY IN THE ELDERLY CARDIAC PATIENT.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

VALIUM®
(diazepam) IV

2-MG TABLETS B.I.D. OR T.I.D.

ROCHE

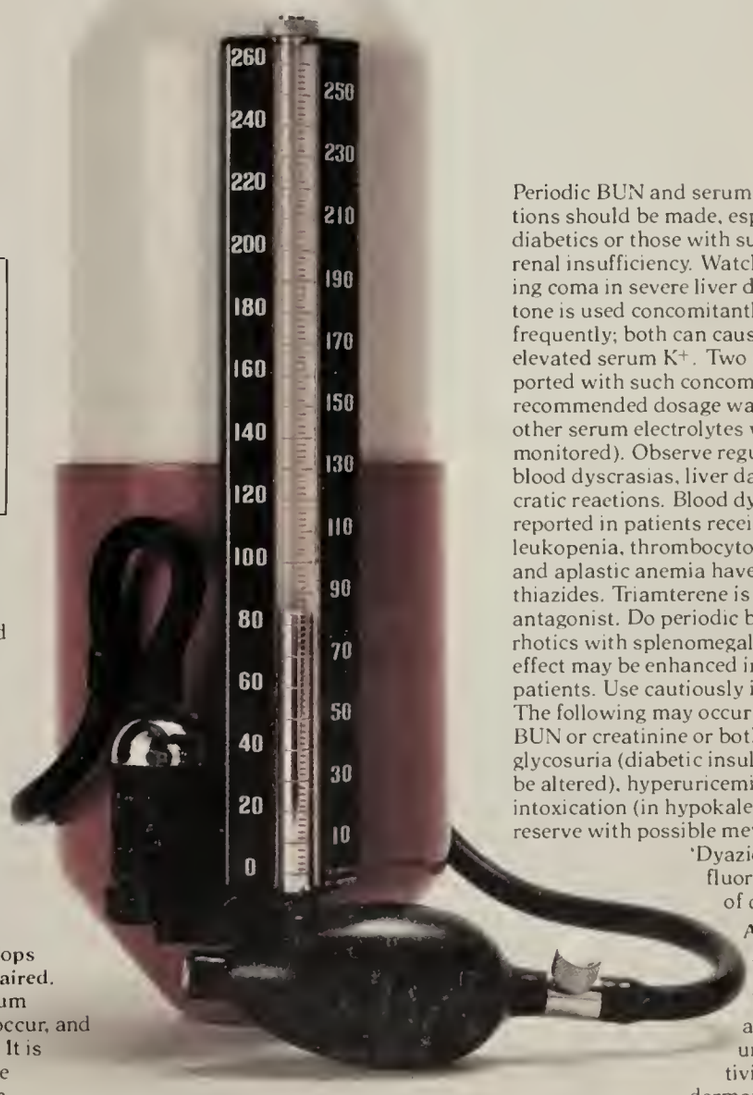
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TRIAMTERENE CONSERVES POTASSIUM WHILE HYDROCHLOROTHIAZIDE LOWERS BLOOD PRESSURE

DYAZIDE®

Each capsule contains 50 mg. of Dyrenium® (triamterene, SK&F Co.) and 25 mg. of hydrochlorothiazide.

MAKES SENSE



Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. A brief summary follows:

* **Warning**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can causc K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

Supplied: Bottles of 100 and 1000 capsules; Single Unit Packages of 100 (intended for institutional use only).

FOR LONG-TERM CONTROL OF HYPERTENSION.* SERUM K^+ AND BUN SHOULD BE CHECKED PERIODICALLY. (SEE WARNINGS SECTION.)

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BURROUGHS WELLCOME CO. MAKES CODEINE COMBINATION PRODUCTS.

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EMPIRIN® COMPOUND c̄ CODEINE #3

Each tablet contains:
codeine phosphate, 32 mg (gr ½),
(Warning: May be habit-forming);
aspirin, 227 mg; phenacetin, 162 mg;
and caffeine, 32 mg.

C III

EMPRACET™ c̄ CODEINE #3

Each tablet contains:
codeine phosphate, 30 mg (gr ½),
(Warning: May be habit-forming);
and acetaminophen 300 mg.

C III

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

INICAL RECOGNITION AND
NAGEMENT OF HEART DISEASE 1978
rch 23-25, 1978. Auditorium, AHSC, cson, AZ. Sponsor: Dept of Cardiology, d Internal Medicine, U of A Health ences Center. Contact: Frank I. Mar-s, M.D., 1501 N. Campbell, Tucson, . Approved for 20 required hours ward the ArMA Certificate in Continuing dical Education.

h ANNUAL ARIZONA
EST SYMPOSIUM
rch 31, April 1-2, 1978. Doubletree Inn, cson, AZ. Contact: Mrs. Linda Alpert, N., Symposium Coordinator, P.O. Box 67, Tucson, AZ 85733.

APRIL

SUES IN CRITICAL CARE MEDICINE
ril 8, 1977. Arizona State University. onsor: Desert Samaritan Hospital, Con-ct: L. A. Rosati, M.D., Dept. of Path., sert Samaritan Hospital. Approved for 7 quired hours toward the ArMA Certifi-te in Continuing Medical Education.

ERIATRIC POW-WOW
ril 7, 8, 9, 1978. Arizona Inn, Tucson, Z. Sponsor: Continuing Medical Educa-n, U of A. Contact: A. Vuturo, M.D. ffice of Continuing Medical Education d Outreach, U of A Health Sciences nter, Tucson, AZ 85724. Approved for required hours toward the ArMA rtificate in Continuing MedicEl Educa-n.

EDIATRIC RADIOLOGY FOR THE
ENERAL RADIOLOGIST
ril 14-16, 1978, Doubletree Inn at cottsdale Mall, Scottsdale, AZ. Sponsor: aricopa County General Hospital. Con-ct: A.R. Sandrock, M.D., Chairman, ept. of Radiology, Maricopa County eneral Hospital, 2601 E. Roosevelt, hoenix, AZ 85008. Approved for 15 quired hours toward the ArMA Certifi-te in Continuing Medical Education.

RIZONA MEDICAL ASSOCIATION 86th
NNUAL CONVENTION.
ril 25-29, 1978, Tucson Community nter and Marriott Hotel, Tucson, AZ. onsor: Arizona Medical Association, c. Contact: Mr. Bruce E. Robinson, 810 . Bethany Home Rd. Phoenix, AZ 85013. pproved for required hours toward the rMA Certificate in Continuing Medical ducation. Detailed program will appear the March issue of *Arizona Medicine*.

MAY

PDATE: PRIMARY CARE 1978
May 3-6, 1978. Arizona Health Sciences Center Sponsor: U of A Continuing Medical Education. Contact: A. Vuturo, M.D., Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 25 required hours toward the ArMA Certificate in Continuing Medical Education.

JUNE

MEDICAL HORIZONS: ADVANCES IN THERAPEUTICS
June 30-July 2, 1978. Little America Hotel, Flagstaff, AZ. Sponsor: Office of Continuing Medical Education, U of A Health Sciences Center, Tucson, AZ. Contact: Anthony F. Vuturo, M.D., AHSC, Tucson, AZ 85724. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

JULY

21st ANNUAL RUIDOSO FAMILY PRACTICE SEMINARO
July 17-20, 1978. Headquarters Inn, of the Mountain Gods at Mescalero, New Mexico. Sponsors: New Mexico Chapter of the American Academy of Family Physicians. Contact: Bob Reid, Convention Dir., P. O. Box 456, Sunland Park, N.M. 88063. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MONTHLY OR WEEKLY

HEADACHE ROUNDS
Tuesday afternoons or Thursday mornings weekly. Sponsor: Headache Group of the Southwest. Contact: Peggy Voigt, 2610 W. Bethany Home Rd., Phoenix, AZ 85017, or phone 242-6646.

SHRINE MEDICS MEETING
Second Tuesday of each month, Doctors Hospital, 5:45 p.m., J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

OFFICE PSYCHIATRY FOR THE PRIMARY PROVIDER
2nd Monday of month, 4811 N 7th Street, Phoenix, AZ Sponsor Arizona Health Plan Contact T R Bittker, M.D , Box 5000, Phoenix, AZ 85010 Approved for required hours toward the ArMA Certificate in Continuing Medical

COUNTER TRANSFERENCE GROUP
Weekly, Thurs. 8-10 p.m. Sponsor. Phoenix Psychiatric Council. Contact. James E. Campbell, M.D., 5051 N 34th St., Phoenix, AZ 85018. Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

MONTLHY MEETING OF TUCSON RADIOLOGISTS
Last Tues. of Month, Plaza International, Tucson, AZ. Sponsor U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., Arizona Medical Center, Dept. of Radiology, Tucson, AZ 85724 Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

REVIEW OF FORENSIC PATHOLOGY, CURRENT CASE, SPECIAL TOPICS
Thursday, weekly. 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists, Contact: H.H. Karnit Schinig, M.D., 120 S. 6th Ave. Phoenix, AZ. Approved for 1 required hour per session toward ArMA CME Certificate.

Arizona State Hospital

STAFF EDUCATION CONFERENCE
Wednesdays, Weekly, 1 p.m. Arizona State Hospital, Phoenix, AZ Sponsor Arizona State Hospital. Contact. Howard E. Wulsin, M.D , Arizona State Hospital, 2500 E. Van Buren, Phoenix, AZ 85008. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

Baptist Hospital

SURGICAL GRAND ROUNDS
4TH TUESDAY OF EACH MONTH
Hospital Auditorium, Baptist Hospital, Phoenix. Sponsor Baptist Hospital Phoenix. Contact. James B. Shields, M.D., 6036 N 19th Ave., Phoenix, AZ 85015. Approved for 1-1/2 required hours per month toward the ArMA Certificate in Continuing Medical Education.

Camelback Hospital

PATIENT STAFFING CONFERENCE
Three times weekly. Camelback Hospital Phoenix, AZ. Sponsor: Camelback Hospital. Contact Medical Director, Camelback Hospital, 5055 N 34th St., Phoenix, AZ 85018. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

Good Samaritan Hospital

DESERT SAMARITAN HOSPITAL
Wednesday Evenings 7 p.m. Sponsor. Desert Samaritan Hospital. Contact: L. A Rosati, M.D , Laboratory, Desert Samaritan Hospital, Mesa, AZ 85202. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

CLINICAL CANCER CONFERENCE
3rd Wednesday every month, Butler Bldg. Conference Room. Good Samaritan Hospital, Phoenix, AZ. Sponsor Good Samaritan Hospital. Contact. John A Bruner, M.D., 926 E. McDowell Road, Phoenix, AZ 85006. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

PULMONARY DISEASE GRAND ROUNDS
Mondays—12 Noon. D-5 North Conference Rm , Good Samaritan Hospital, Phoenix, AZ. Sponsor Pulmonary Disease Teaching Service, Good Samaritan Hospital. Contact Bernard E. Levine, M.D., Pulmonary Function Laboratory, Good Samaritan Hospital, 1033 E. McDowell Hospital, Phoenix, AZ 85006. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

Phoenix Indian Hospital

MONTHLY MEDICAL EDUCATION SEMINAR
Third Monday of the Month, 6 30 p.m., Kiva Conference Room, Phoenix Memorial Hospital, Sponsor: Medical Staff of Memorial Hospital. Contact George Scharf, M.D., 1201 South 7th Avenue, Phoenix, AZ 85007 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PEDIATRIC GRAND ROUNDS
Tuesday 7:30 - 8:30 a.m. in Phoenix: 1st Tue. - Phoenix Indian Hospital, 2nd Tue. - Maricopa County Hospital, 3rd Tue. - Good Samaritan Hospital, 4th Tue. - St. Joseph's Hospital
Sponsor: Phoenix Hospital's Affiliated Pediatric Program. Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

MONTHLY MEDICAL EDUCATION SEMINAR
Third Monday of the Month, 6:30 p.m., Kiva Conference Room, Phoenix Memorial Hospital. Sponsor: Medical Staff of Memorial Hospital. Contact: George Scharf, M.D., 1201 South 7th Avenue, Phoenix, AZ 85007. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

TUMOR BOARD CONFERENCE
Every Friday at Noon, Kiva Conference Room, Phoenix Memorial Hospital. Sponsor: Phoenix Memorial Hospital. Contact. Howard Kimball, M.D., 333 West Thomas Road, Phoenix, AZ 85013. Approved for credit toward the ArMA Certificate in Continuing Medical Education.

Scottsdale memorial Hospital

CLINICAL PATHOLOGICAL CONFERENCE
4th Monday, Monthly, 12 45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ. Sponsor: Medical Staff. Contact: R. C. Good, M.D., Director of Medical Education, 7300 E. 4th St., Scottsdale, AZ. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

FAMILY PRACTICE CONFERENCE
1st Monday, Monthly, 12:45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ. Sponsor: Medical Staff. Contact: R. C. Good, M.D., Dir. of Medical Education, 7300 E. 4th St., Scottsdale, AZ. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

MEDICAL GRAND ROUNDS
3rd Monday, Monthly, 12:45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ. Sponsor: Medical Staff. Contact: R. C. Good, M.D., Dir. of Medical Education, 7300 E. 4th St., Scottsdale, AZ. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

MORBIDITY & MORALITY CONFERENCE
2nd Monday, Monthly, 12:45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ. Sponsor: Medical Staff. Contact. R C. Good, M.D., Dir. Medical Education, 7300 E. 4th St., Scottsdale, AZ. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

CARDIOLOGY CONFERENCE
Weekly—Friday 8-9 a.m., St. Mary's Hospital Auditorium, Tucson, AZ. Sponsor: St. Mary's Hospital. Contact: A. L. Forte, M.D., St. Mary's Hospital, Tucson, AZ 85724. Approved for one required hour toward the ArMA Certificate in Continuing Medical Education.

St. Mary's Hospital, Tucson

GRAND ROUNDS
Each Thursday 7 a.m.-8 a.m., St. Mary's Hospital and Health Center, Sponsor Depts. of Medicine, Surgery, Radiology, Pathology and Family Practice. Contact: Richard Silver, M.D., Chairman, Medical Education and Library Committee, Century Medical Plaza, Suite 160, 1701 West St. Mary's Road, Tucson, AZ 85703. Approved for 1 required hour per round toward the ArMA Certificate in Continuing Medical Education.

U of A Health Sciences Center

CLINICAL IMMUNOLOGY, ALLERGY AND RHEUMATOLOGY ROUNDS
Every Friday Noon-1 p.m. Sponsor U. of A. College of Medicine, Dept. of Internal Medicine, Clinical Immunology Section. Contact: John Boyer, M.D., U. of A. College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

CEREBROVASCULAR DISEASE CONFERENCE
Mondays 5-6 p.m., weekly, Rm 5505 Surgery, U of AHSC. Sponsor: Depts. of Surgery, Neurology and Radiology, UAHSC. Contact: Dr. Wesley Moore, U of A Health Sciences Center, Tucson, AZ 85724.

ENDOCRINOLOGY SEMINAR
Every Thursday, Noon-1 p.m., 1st, 3rd & 5th Thursday—Rm. N318, VA Hospital, 2nd & 4th Thursday, Rm. 6505, Tucson Medical Center, Sponsor. U of A. College of Medicine, Department of Internal Medicine, Department of Internal Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

HEMATOLOGY-ONOCOLOGY CLINICAL CONFERENCE
Every Tuesday, Noon-1 p.m. 1st, 3rd & 5th Tuesdays—Rm. 6505, AZ Medical Center. 2nd & 4th Tuesdays—Rm. N318, Veterans Adm. Hospital. Sponsor: U. of A. College of Medicine, Dept of Internal Medicine. Contact: Sidney Salmon, M.D., U. of A. College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

MEDICAL GRAND ROUNDS
Every Wednesday, Noon-1 p.m. 1st, 3rd & Wednesday—Staff Conf Rm., VA Hospital. & 4th Wednesday—Rm. 5403, Arizona Medi Center. Sponsor U. of A College of Medic Dept. of Internal Medicine. Contact. Jay Sm M.D., U. of A. College of Medicine, Tucson, 85721 Approved for 1 required hour per s sion toward the ArMA Certificate in Continu Medical Education.

NUCLEAR MEDICINE
Thursday, 7:30 a.m., Radiology Co Rm., AHSC, Tucson. Sponsor: Dept. Diagnostic Radiology. Contact: Irwin Freundlich, U of A Health Scienc Center, Tucson, AZ 84724. Approved one required hour per session towa ArMA CME Certificate.

NEUROLOGY GRAND ROUNDS
Every Friday, 12 noon. 1st, 3rd & Fridays, U of A Health Sciences Cent Rm. 7505. 2nd and 4th Fridays, Vetera Administration Hospital. Sponsor: U of Health Sciences Center, Dept. of Neur ogy. Contact: William A. Sibley, M.I Dept. of Neurology U of A Hea Sciences Center, Tucson, AZ 85724. A proved for 1 required hour per sessi toward the ArMA Certificate in Continui Medical Education.

NEUROPATHOLOGY REVIEW CONFERENCE
Thursdays, 8:30 a.m., Rm 5120 Arizo Health Sciences Center. Sponsor: U of College of Medicine. Contact: Peter Johnson, M.D., 1501 N. Campbell Av Tucson, AZ 85724. Approved for 1 quired hour per session toward the Ar Certificate in Continuing Medical Edu tion.

NEUROSCIENCES SEMINAR
Every Friday, 7:30 a.m. U of A Hea Sciences Center, Rm. 7505, Tucson, A Sponsor: Dept. of Neurology. Conta Colin R. Bamford, M.D. & Jose Laguna, M.D., Dept. of Neurology, U of Health Sciences Center, Tucson, A 85724. Approved for 1 required hour p session toward the ArMA Certificate Continuing Medical Education.

PATHOLOGY SEMINAR
Friday, 4:30-5:30 p.m., Rm 5120 AHS Tucson, AZ. Sponsor: Dept. of Patholo Contact: Dr. Kenneth Ryan, Dept. of Pa U of A Health Sciences Center, Tucs AZ 85724. Approved for one required h per session toward the ArMA Continui Medical Education Certificate.

SON PATHOLOGIST IFERENCE
t Monday of the month, 7:30-9 p.m., ation Between Tucson Pathology f. Rms., Sponsor: Tucson Patholo-s. Contact: Dr. Merle Clard, U of A lth Sciences Center, Dept. of Path., son, AZ 84724. Approved for 1 re-ed hour per session toward the ArMA ificate in CME.

IATRIC GRAND ROUNDS
y Tuesday, 12:30 p.m. - 1:30 p.m. Al-ates between Tucson Medical Clinic U of A Health Sciences Center. Spon-U of A College of Medicine, Dept. of atrics. Contact: Hugh Thompson, ., 1501 N. Campbell, Tucson, AZ. roved for 1 required hour per session ard the ArMA Certificate in Continuing ical Education.

BLEM CASE WORKSHOPS
Monday of each month 7.30 a.m. Room), Arizona Medical Center, Tucson, AZ. nsor: Division of Ophthalmology, U. of A. ege of Medicine. Contact: H. E. Cross, M.D., ., Arizona Medical Center, Dept of Sur-, Tucson, AZ. Approved for 2 required s toward the ArMA Certificate in Continu-Medical Education

CHIATRIC GRAND ROUNDS
y Wed., Sept to May, 4-5:30 p.m. Rm. 8403, ona Medical Center, Tucson, AZ. Sponsor A College of Medicine Dept. of Psychia-Contact: Alan Levenson, M.D., U of A. ege of Medicine, Tucson, AZ 85721. Ap-ed for 1-1/2 required hour per session rd the ArMA Certificate in Continuing ical Education

EST RADIOLOGY
day, 5-6 p.m., weekly, Rm. 1535F, C, Tucson, AZ. Sponsor: Dept. of iology. Contact: Irwin M. Freundlich, ., U of A Health Sciences Center, t. of Radiology, Tucson, AZ 85724. roved for 1 required hour per session ard ArME CME Certificate.

STROINTESTINAL RADIOLOGY
ndays, 7:30 a.m., weekly except for mer months. Sponsor: Dept. of Radi-gy. Contact: Irwin M. Freundlich, ., Diagnostic Radiology, U of A lth Sciences Center, Tucson, AZ 24. Approved for 1 required hour ard ArMA CME Certificate.

DIATION ONCOLOGY ANNING CONFERENCE
day, weekly, 8:30-10 a.m., AHSC Rm. 5. Sponsor: Radiation oncology tion, UAHSC, Tucson, AZ. Contact: bert S. Heusinkveld, M.D., Ph.D., U of Health Sciences Center, Tucson, AZ 24. Approved for 1.5 required hours session toward ArMA Certificate in E.

RADIOLOGY-RHEUMATOLOGY CONFERENCE
Thursday, 8 a.m., UAHSC, Library, Rm. 1535C. Sponsor: Dept. of Radiology, Contact: Irwin M. Freundlich, U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour toward the ArMA Certificate in CME.

VASCULAR RADIOLOGY
January thru June 1978. 7:30 a.m. to 8:30 a.m. every Friday. Dept. of Radiology. Sponsor: Dept. of Radiology, U of A Health Sciences Center. Contact: I. M. Freundlich, M.D., U of A Health Sciences Center Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

RADIOLOGY INTERESTING CASE CONFERENCE
Thursday, noon, Veterans Administration Hospital, Tucson, AZ. Sponsor: U of A Health Sciences Center, Tucson, Radiology Dept. Contact: Irwin M. Freundlich, M.D., U of A health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA CME Certificate.

RESIDENT'S CONFERENCE
Wednesday/weekly 5-6 p.m., Diag. Radiology Conf. Rm., U of A Health Sciences Center. Sponsor: U of A Health Sciences Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour per session toward the ArMA Certificate in Continuing Medical Education.

TRAUMA CONFERENCE
Every Thursday, 4-5:00 p.m. Health Sciences Center, Rm. 5505, Tucson, AZ. Sponsor: U of A College of Medicine, Dept. of Surgery. Contact: Martin Silverstein, M.D., Dept. of Surgery, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

GRAND WARD ROUNDS – TRAUMA
Every Tuesday, 8 a.m. Arizona Medical Center, Tucson, AZ. Sponsor U. of A. College of Medicine, Surgery Dept., Trauma Section. Contact Martin Silverstein, M.D., U of A. College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

MONTHLY MEETING TUCSON ULTRASONOGRAPHY GROUP
First Wednesday of month, 4:30-5:30 p.m., Arizona Health Sciences Center. Sponsor: Dept. of Diagnostic Radiology. Contact: Irwin M. Freundlich, M.D., AHSC, Tucson, AZ 85724. Approved for 1 required hour per session toward ArMA CME Certificate.

TUCSON UROLOGICAL SOCIETY SCIENTIFIC MEETING
Monthly, Sponsor: Depts of Surgery & Urology, AHSC, Tucson. Contact: George W. Drach, M.D., U of A ealth Sciences Center, Tucson, AZ 85724. Approved for 1.5 required hours per session toward ArMA CME Certificate.

GENERAL UROLOGY CONFERENCE
Tuesday 5-6 p.m., Thursday noon-1:30 p.m. AHSC Meeting rms., & VA Hospital. Sponsor: Dept of Surgery/Urology Section. Contact: George W. Drach, M.D. Dept. of Surgery/Urology Section, U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour toward ArMA CME Certificate.

VA Hospital, Phoenix

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Each Wednesday, 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact: Jasper L. McPhail, M.D., VA Hospital 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

CARDIOLOGY CONFERENCE
2nd Thursday 1 p.m. T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd. Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

MEDICAL-SURGICAL CHEST CONFERENCE
1st and 3rd Thursday, 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact: Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

CLINICOPATHOLOGY CONFERENCE
4th Thursday of 3rd Mo. (Mar., June., Sept. & Dec.), 1 p.m., T-5. Sponsor: VA Hospital Phoenix. Contact: Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Education.

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HEPATOLOGY CONFERENCE
2nd and 4th Tuesday, 1 p.m., 2128. Sponsor VA Hospital Phoenix. Contact. Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

GASTROENTEROLOGY CONFERENCE
1st and 3rd Tuesday, 1 p.m., T-5. Sponsor: VA Hospital Phoenix, Contact: Jasper L. McPhail, M.D. VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

GI CONFERENCE
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G.I.-RADIOLOGY CLINICAL CORRELATION CONFERENCE
Each Tuesday, 1 p.m., T-5. Sponsor: VA Hospital Phoenix. Contact: Jasper L. McPhail, M.D., Veterans Administration Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL SURGICAL G.I. CONFERENCE
1st & 3rd Friday - 2 p.m. -2128 Sponsor: VA Hospital, Phoenix. Contact: Jasper L. McPhail, M.D., VA Hospital, 7th Ave. & Indian School Rd., Phoenix. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL SERVICE GRAND ROUNDS
Each Friday, 11 a.m., T-5 Sponsor: VA Hospital Phoenix. Contact. Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

SURGERY-PATHOLOGY CONFERENCE
Each Thursday, 7 a.m., 2128 Sponsor. VA Hospital Phoenix Contact: Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

UROLOGY-PATHOLOGY CONFERENCE
Each Wednesday, 7 a.m., 2128. Sponsor: VA Hospital Phoenix. Contact Jasper L. McPhail, M.D., VA Hospital, 7th and Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

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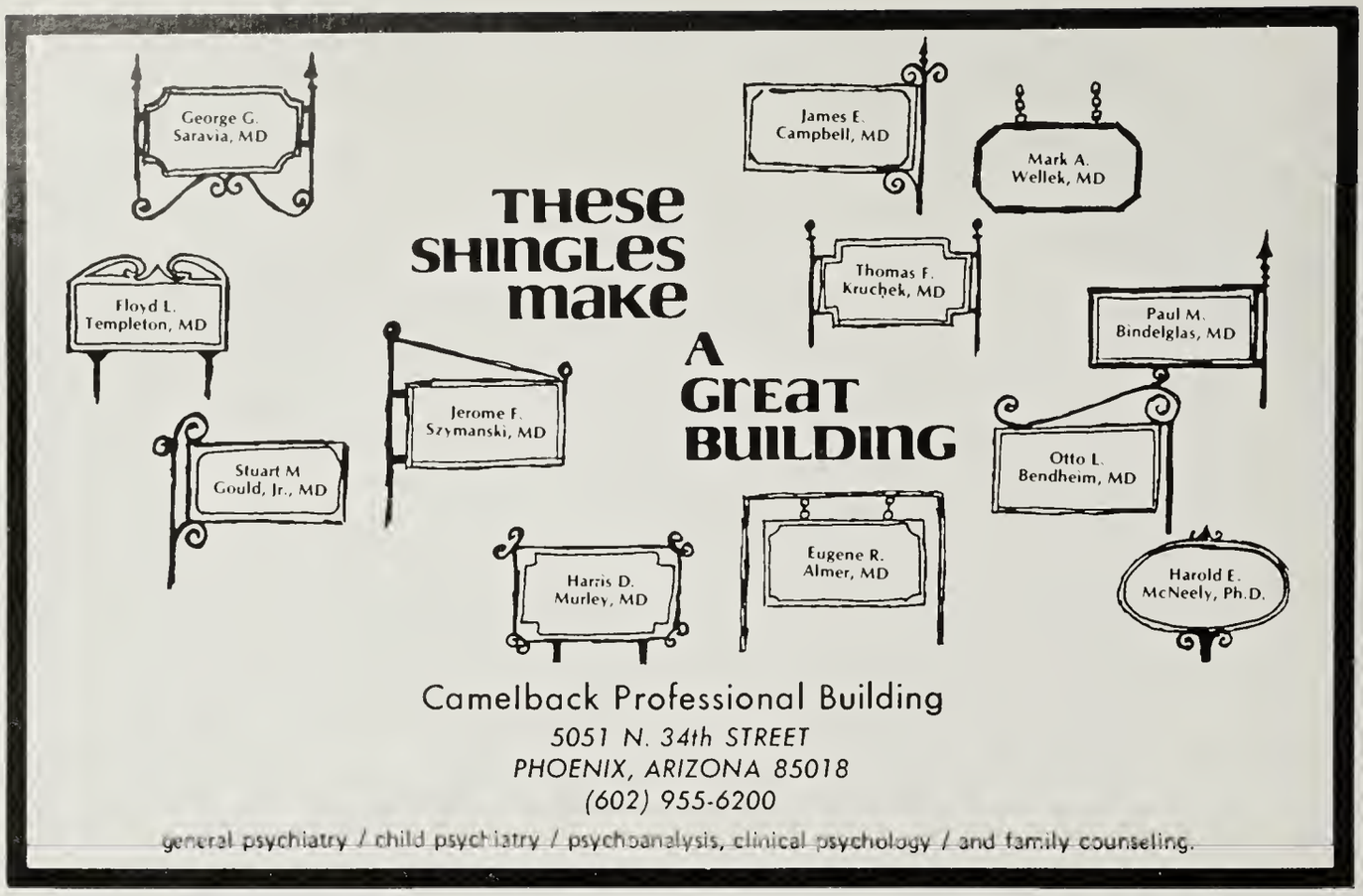
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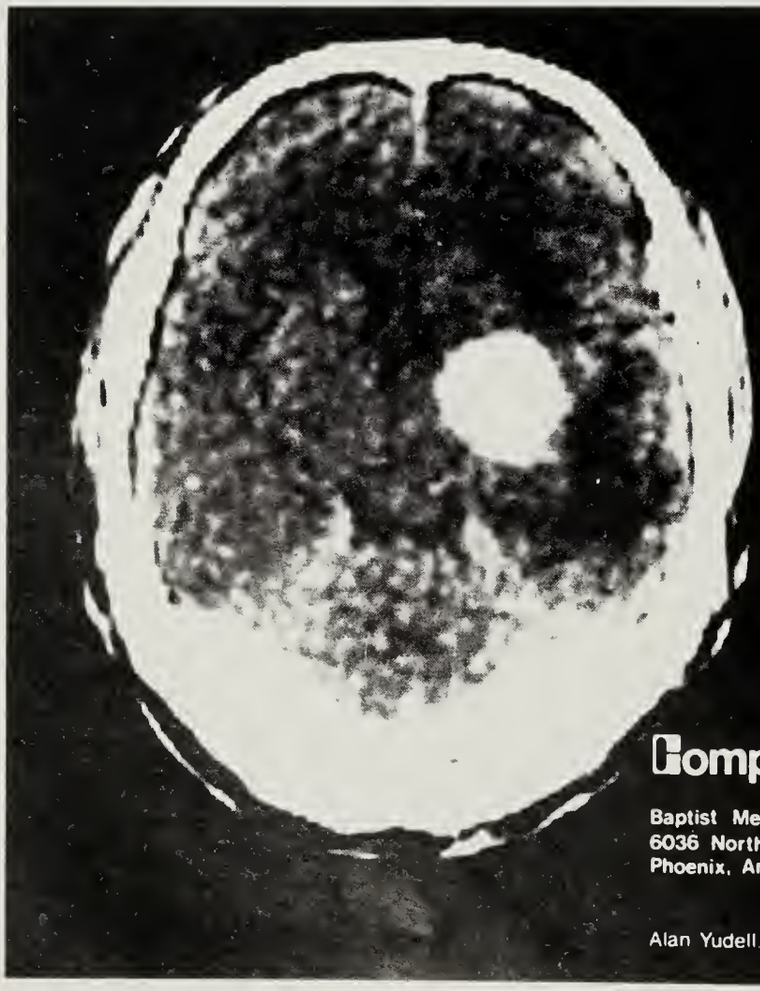
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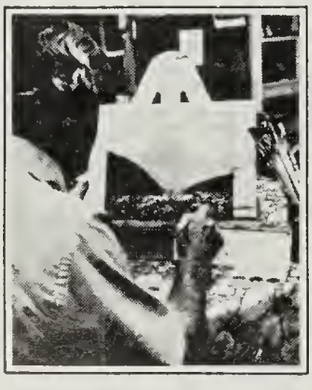
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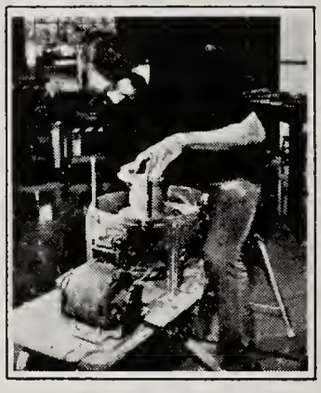
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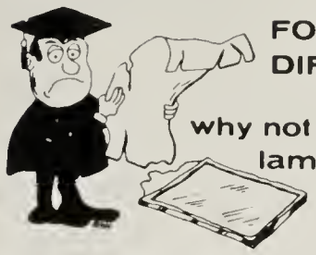
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L. XXXV NO. 2

A character all its own.



Valium (diazepam) is a benzodiazepine with a character all its own. Pharmacologically, it has been described as more potent mg-per-mg than other available anxiolytic benzodiazepines. Pharmacokinetically, only Valium provides active *diazepam* as well as the active metabolites 3-hydroxydiazepam, desmethyldiazepam and oxazepam.

But the individual character of Valium is even more apparent clinically than pharmacokinetically. And far more significant. That's because of the patient response obtained with Valium. A response which brings a calmer frame of mind. A response which has a pronounced effect on the somatic symptoms of anxiety, particularly muscular tension. A response which helps the patient feel more like himself again because of the way Valium reduces the overwhelming symptoms of anxiety and psychic tension.

Another important aspect of the clinical character of Valium is safety. Though drowsiness, ataxia and fatigue are possible, these and more serious side effects are rarely a problem. Of course, as with all CNS-acting drugs, patients taking Valium should be cautioned against driving, operating dangerous machinery or the simultaneous ingestion of alcohol.

Unquestionably, many psychotherapeutic agents, including other benzodiazepines, have antianxiety effects. But one fact remains: you get a certain kind of patient response with Valium. It's a response you want. A response you know. A response you trust as part of your overall management of anxiety and psychic tension.

Valium® (diazepam) IV

2-mg, 5-mg, 10-mg scored tablets

a prudent choice in psychic tension and anxiety

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication, abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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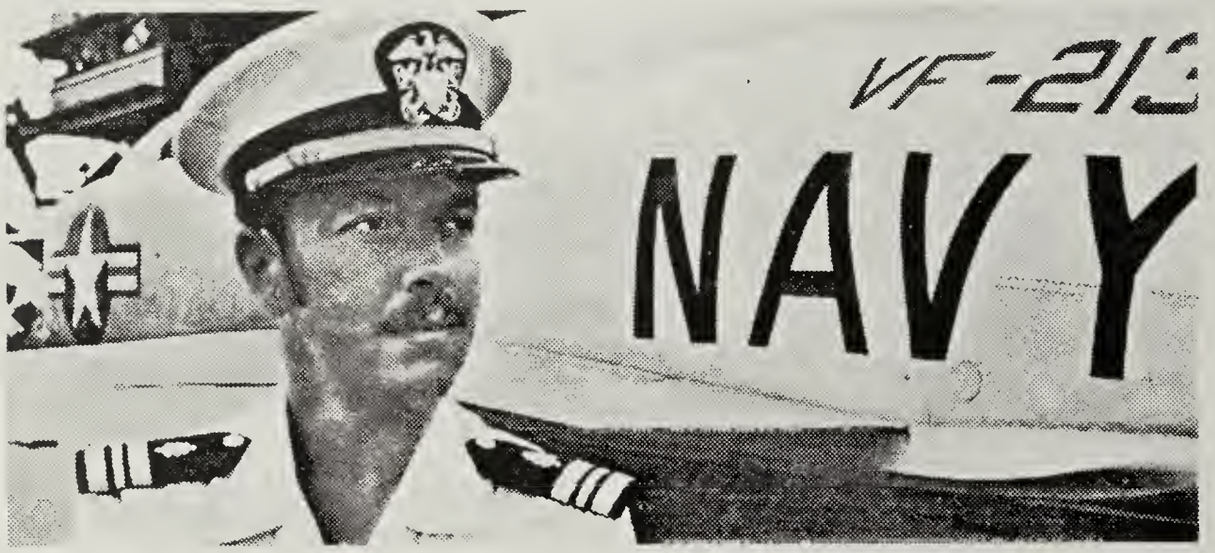
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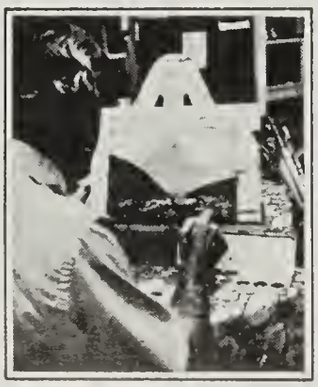
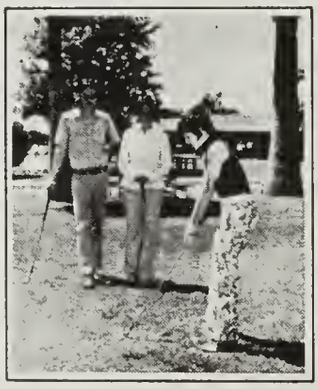
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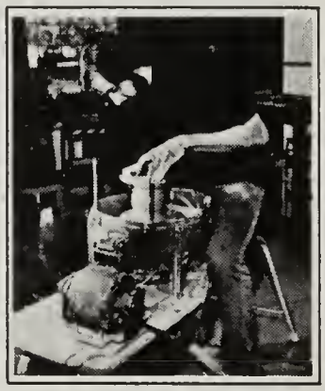
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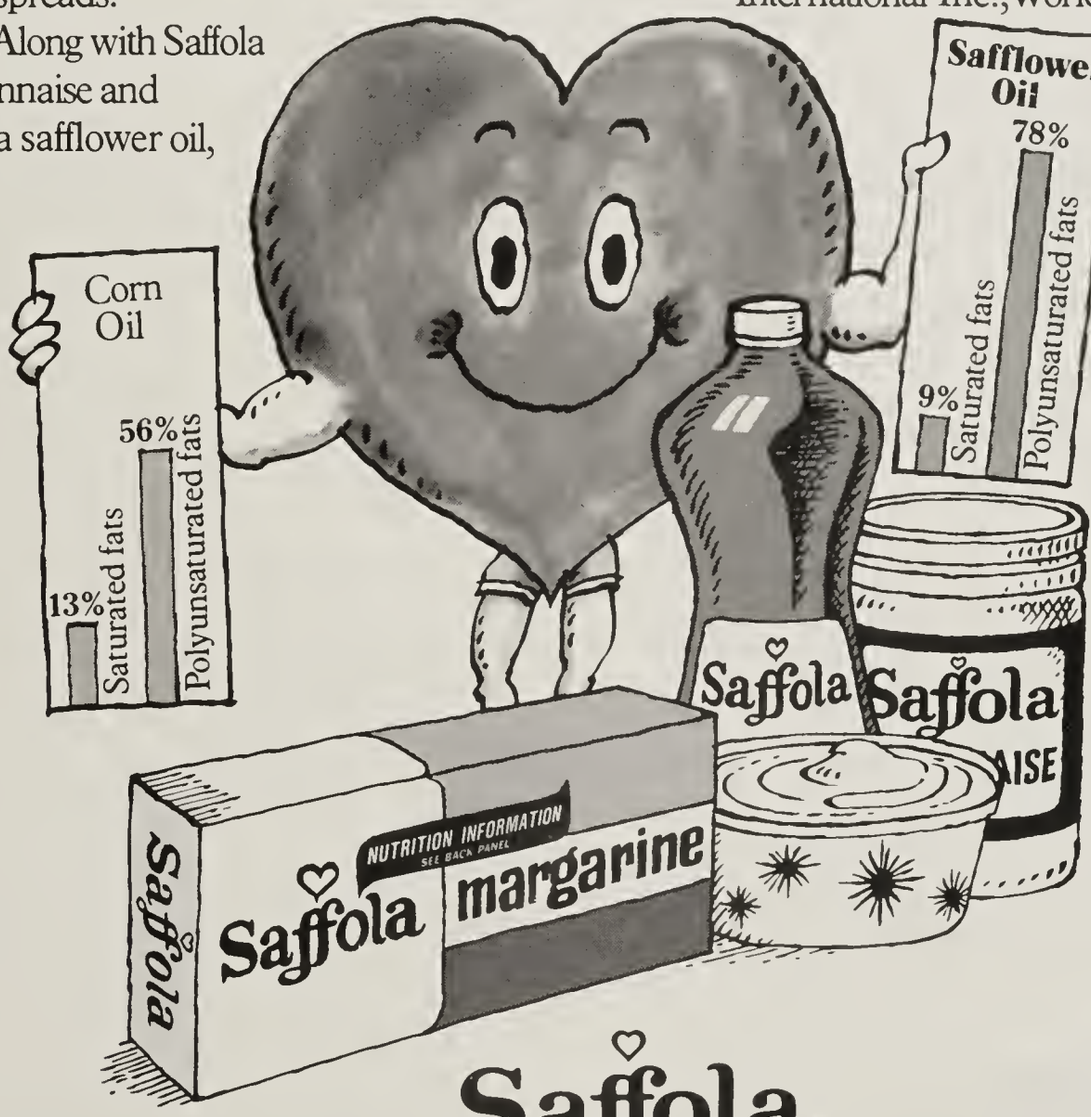
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When pain complicates acute cystitis*

Azo Gantanol®

Each tablet contains 0.5 Gm sulfamethoxazole and 100 mg phenazopyridine HCl

for the pain for the pathogens



□ **Early relief of painful symptoms** such as burning and pain associated with urgency and frequency.

□ **Effective control of susceptible pathogens** such as *E. coli, Klebsiella-Aerobacter, Staph. aureus, Proteus mirabilis* and, less frequently, *Proteus vulgaris.*

□ **Appropriate antibacterial therapy:** up to three days therapy with Azo Gantanol, then 11 days with Gantanol® (sulfamethoxazole), 0.5 Gm tablets.

*Nonobstructed; due to susceptible organisms

Before prescribing, please consult complete product information, a summary of which follows:

Indications: In adults, urinary tract infections complicated by pain (primarily pyelonephritis, pyelitis and cystitis) due to susceptible organisms (usually *E. coli, Klebsiella-Aerobacter, Staphylococcus aureus, Proteus mirabilis* and, less frequently, *Proteus vulgaris*) in the absence of obstructive uropathy or foreign bodies.

***Note:* Carefully coordinate *in vitro* sulfonamide sensitivity tests with bacteriologic and clinical response; add aminobenzoic acid to follow-up culture media. The increasing frequency of resistant organisms limits the usefulness of antibacterials including sulfonamides.** Measure sulfonamide blood levels as variations may occur; 20 mg/100 ml should be maximum total level.

Contraindications: Children below age 12; sulfonamide hypersensitivity; pregnancy at term and during nursing period; because Azo Gantanol contains phenazopyridine hydrochloride it is contraindicated in glomerulonephritis, severe hepatitis, uremia, and pyelonephritis of pregnancy with G.I. disturbances.

Warnings: Safety during pregnancy not established. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been reported and early clinical signs (sore throat, fever, pallor, purpura or jaundice) may indicate serious blood disorders. Frequent CBC and urinalysis with microscopic examination are recommended during sulfonamide therapy.

Precautions: Use cautiously in patients with impaired renal or hepatic function, severe allergy, bronchial asthma; in glucose-6-phosphate dehydrogenase-deficient individuals in whom dose-related hemolysis may occur. Maintain adequate fluid intake to prevent crystalluria and stone formation.

Adverse Reactions: *Blood dyscrasias* (agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia); *allergic reactions* (erythema multiforme, skin eruptions, Stevens-Johnson syndrome, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis); *G.I. reactions* (nausea, emesis, abdominal pains, hepatitis, diarrhea, anorexia, pancreatitis and stomatitis); *CNS reactions* (headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo and insomnia); *miscellaneous reactions* (drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon). Due to certain chemical similarities with some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia. Cross-sensitivity with these agents may exist.

Dosage: Azo Gantanol is intended for the acute, painful phase of urinary tract infections. *Usual adult dosage:* 2 Gm (4 tabs) initially, then 1 Gm (2 tabs) B.I.D. for up to 3 days. If pain persists, causes other than infection should be sought. After relief of pain has been obtained, continued treatment with Gantanol (sulfamethoxazole) may be considered.

Note: Patients should be told that the orange-red dye (phenazopyridine HCl) will color the urine.

Supplied: Tablets, red, film-coated, each containing 0.5 Gm sulfamethoxazole and 100 mg phenazopyridine HCl—bottles of 100 and 500.

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Nutley, New Jersey 07110

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DYAZIDE®

Each capsule contains 50 mg. of Dyrenium® (triamterene, SK&F Co.) and 25 mg. of hydrochlorothiazide.

MAKES SENSE



Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. A brief summary follows:

* **Warning**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. Associated **widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

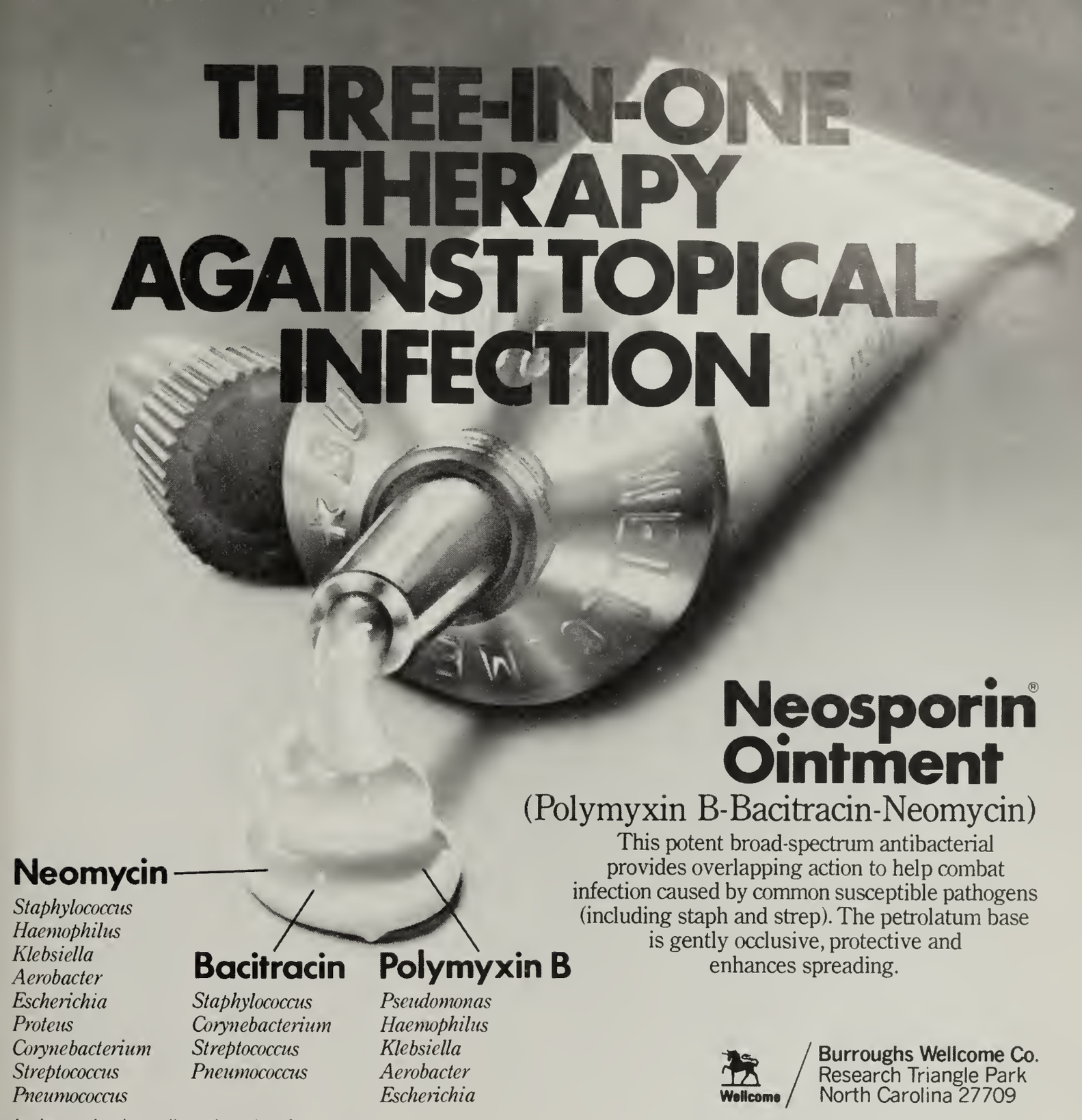
Supplied: Bottles of 100 and 1000 capsules; Single Unit Packages of 100 (intended for institutional use only).

FOR LONG-TERM CONTROL OF HYPERTENSION.* SERUM K^+ AND BUN SHOULD BE CHECKED PERIODICALLY. (SEE WARNINGS SECTION.)

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Neosporin® Ointment

(Polymyxin B-Bacitracin-Neomycin)

This potent broad-spectrum antibacterial provides overlapping action to help combat infection caused by common susceptible pathogens (including staph and strep). The petrolatum base is gently occlusive, protective and enhances spreading.

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Proteus
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Pneumococcus

Bacitracin

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Corynebacterium
Streptococcus
Pneumococcus

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In vitro overlapping antibacterial action of Neosporin® Ointment (polymyxin B-bacitracin-neomycin).

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Neosporin® Ointment

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Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 units; zinc bacitracin 400 units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML

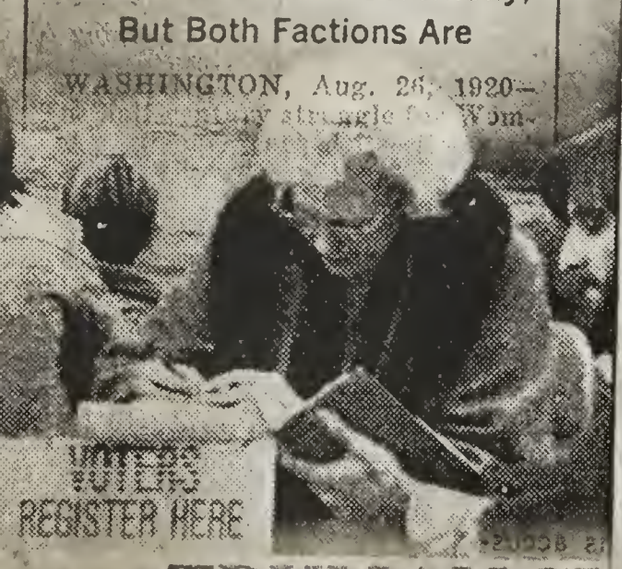
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Social Security Bill Is Signed; Gives Pensions to Aged, Jobl

Roosevelt Approves Message Intended to Benefit 30,000 Persons When States Adopt Cooperating Laws–He Co the Measure 'Cornerstone' of His Economic Program

WASHINGTON, Aug. 14, The Social Security Bill, pro a broad program of unemplo insurance and old age pen and counted upon to benefit 20,000,000 persons, became la day when it was signed by P dent Roosevelt in the presen those chiefly responsible for ting it through Congress.

Mr. Roosevelt calle the me "the cornerstone in a stru which is being [illegible] me[illegible]s comple[illegible]

COM

SENATE APPROVES 18-YEAR OLD VOTE IN ALL ELECTIONS

Amendment to Constitution is Sent to House, Where Passage is Expected

WASHINGTON, March 10, 1971—The Senate approved today 94 to 0 and sent to [illegible]

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NEW WORLD HOPE

President Hails 'Great Instrument of Peace,' Insists It Be Used

HISTORIC LANDMARK

Meeting Gives Standing Ovation as Executive Pictures Peace Gain

"If we fail to use it," he declared to the solemn final meeting of the delegates, 'we shall betray all of those who have died in order that we might meet here in freedom and safety to create it.'

"If we seek to use it selfishly—for the advantage of any one nation or any small group of nations—we shall be equally guilty of that betrayal."

Fervent Interpolation

The President, speaking in the auditorium of the War Memorial Opera House, built in memory of sons of the Golden Gate city who gave their lives in the first World War, in which he himself served, seemed to give unconscious expression to the solemn feeling of the occasion when, at the outset of his speech, he interpolated the words, half a hope, half a prayer:

"Oh, what a great day this can be in history!"

Just before the plenary session the President accompanied the eight United States delegates

SIGNE the Draf Ends Nov

WASHINGTON, Jan. 27, 1973—"With the signing of the peace agreement in Paris today, and after receiving a report from the Secretary of the Army that



PATIENT PACKAGE INSERTS: A CONCEPT WHOSE TIME HAS COME?

The consumer's right to know is an irreversible and desirable trend of the Seventies. It extends, and properly, to a patient's right to know more about his or her prescription medications. One way, gaining favor, is through patient package inserts. Wisely-prepared and properly distributed when medically indicated, they could markedly improve patient knowledge and drug therapy—laudable goals by anyone's standards.

The PMA endorses these goals and will work with government, the health professions and consumers to achieve them.

The Advantages

The concept holds promise of benefits: better patient understanding of the product prescribed, better adherence to the treatment plan, and more awareness of possible side reactions.

Every doctor has had patients who fail to finish antibiotic regimens because they feel better. Some patients assume that if one tranquilizer or analgesic is good, two may be twice as good. Still others fail to report dizziness while on antihypertensive therapy —and so on.

Problems like these might arise less often if the patient received written information in addition to verbal instructions. Some studies suggest that patients are more receptive to such materials, and they more often understand the verbal instructions and follow them, when inserts are used.

The Disadvantages

There are also some potential problems. Obviously, the inserts must be clearly phrased, without extraneous or complex detail. How much information is enough? How can it be kept current? Should all patients receive the same information? Should inserts be included with all drugs? Should only potential problems be listed or are patients better off with a "fair balance" presentation that describes usefulness as well as drawbacks?

These and similar questions require answers, since model inserts have yet to be properly developed and tested. Despite the need for these studies, the FDA is proceeding prematurely with inserts on selected products. We think the Congress is the only place where the matter can be given the proper legal status and direction, particularly since it represents a conceptual change in the legal, medical and social framework of the nation's prescription drug information system.

The Solution

The PMA believes that carefully-devised pilot studies of various kinds of inserts are needed. They should be developed and implemented with full participation by doctors, pharmacists, consumers, communications experts and the drug industry. Such studies will provide reliable pathways to follow, so that inserts will be useful aids to medical practice.

And particularly we think that you should be closely involved in this debate and in these studies and decisions. Otherwise, people with less experience and qualifications may control the purposes, content and use of a tool with considerable promise for improved patient care. It could make a difference in your practice tomorrow, and more importantly, in the health of your patients.

PMA
THE PHARMACEUTICAL MANUFACTURERS ASSOCIATION
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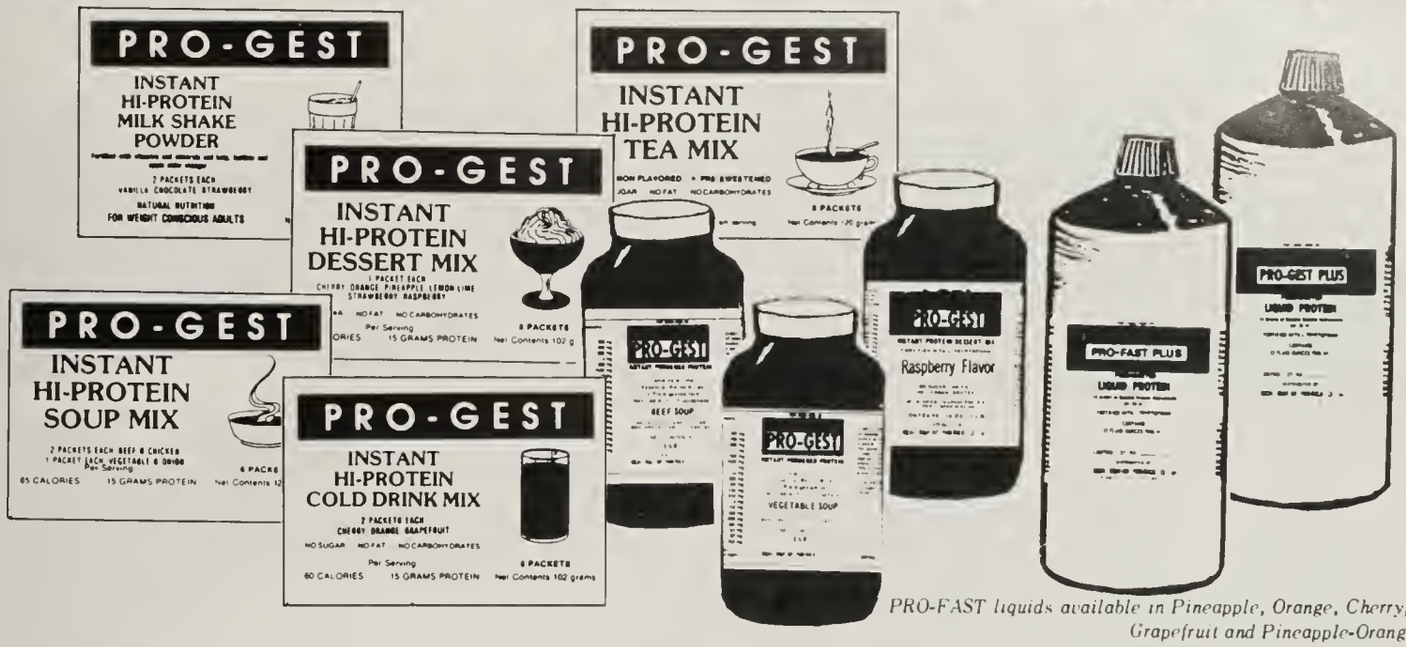
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Current Status Of The Chemotherapy Of Tuberculosis

Part I

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ABSTRACT:

Recent advances in the chemotherapy of tuberculosis have resulted in a dramatic decline in the incidence of this once-dreaded disease. Despite the availability of effective drugs, confusion still exists as to whom to treat, how many drugs to use, and how long to treat patients. The discussion which follows attempts to clarify the management of tuberculosis therapy as well as to explore the newer concepts of short-course therapy and chemoprophylaxis.

INTRODUCTION

Since the 1930's the incidence of tuberculosis per hundred thousand population has declined dramatically. Taken by decade, the greatest declines in the number of new cases have occurred in the decades following the advent of effective chemotherapy.[1] Streptomycin was first used in 1943 and by the early 1950's Isoniazid (INH) was in clinical use. Table I shows the percentage decline by decade and indicates that great strides were made in the rate of reduction of new cases of tuberculosis in the 20-year period 1952-1972. It is apparent that declines in previous decades must have been due to factors other than chemotherapy. A higher standard of living with less crowding in small buildings in urban centers has been a factor. Isolation of newly diagnosed cases has been important in reducing transmission. The development of collapse therapy, including pneumothorax, pneumoperitoneum, thoracoplasty, and later, resectional surgery represented great strides in the management of advanced disease. Effective chemotherapy, however, has been the most notable advance in the treatment of tuberculosis.

Treatment: General Considerations

Before proceeding with a detailed discussion of the pharmacology of the individual tuberculosis drugs, several general considerations about therapy need to be reviewed. The goals of anit-tuberculous therapy should be to provide 1) rapid relief of pulmonary and

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TABLE I

DECLINE IN NEW CASES OF TUBERCULOSIS BY DECADE

Decade	% Decrease
1932-1942	4%
1942-1952	6%
1952-1962*	39%
1962-1972*	37%

**Era of anti-tuberculosis chemotherapy*

systemic symptoms; 2) rapid return of the patient to normal health and subsequently back to work and to his family; and 3) to insure against transmission of the infection.[3] These goals should be met with a minimum of dislocation to the patient, family, home and work environments.

An initial brief period of hospitalization of up to two weeks is warranted for most patients suspected of having active tuberculosis.[2] Since the greatest degree of infectivity exists prior to initiation of chemotherapy, temporary respiratory isolation reduces the risk of transmission while the patient is being evaluated. In addition the hospital setting affords an excellent chance to educate the patient about his disease emphasizing: 1) that the disease is transmitted via droplet nuclei expelled from the lungs, and 2) that in all probability he will be cured of his tuberculosis if he faithfully takes his drugs. prolonged hospitalization is necessary only when the patient is sick enough to require supportive care; when certain hospital based tests are necessary for further diagnostic workup or to monitor therapy; and when isolation cannot be achieved at home, expecially when young children, diabetics, or immunocompromised patients are present in the same household. The past practice of hospitalizing patients in sanitoriums is no longer justifiable on medical or public health grounds.[3]

After the initiation of anti-tuberculous chemotherapy the patient is rapidly rendered non-infectious. One study has estimated that patients are 0.07% as infectious after one month of chemotherapy as they were before treatment.[3,4] It is difficult to apply rules as to when the patient is non-infectious. In most instances, however, decline in malaise, fever, cough, sweats, and bacilli counts in the sputum all serve to indicate that the patient is being effectively treated. These indications usually occur within two weeks after the start of drug treatment.[5] Respiratory isolation can usually be discontinued within two weeks. The use of gowns, special dishes, and disinfectants is of no proven value in preventing transmission.[3] Studies have shown that factors such as rest, diet, climate, and psychological state are not important in determining the outcome of chemotherapy.

First and Second Line Drugs

Anti-tuberculous agents can be divided into two categories: the so-called "first line" and "second line" drugs. These medications are listed in Table II. The drugs in the left column are called "first line" because they are used in initial treatment regimens. Isoniazid, ethambutol, streptomycin and rifampin, employed in two- and three-drug combinations represent the great bulk of drugs used in most cases of tuberculosis. They are less toxic and more easily absorbed than other drugs. PAS, formerly regarded as a first line drug, has been abandoned by all but a few state and county institutions because of its intolerable gastrointestinal side effects. The first line drugs will be discussed in detail along with some helpful reminders in their effective use. The second line drugs will not be discussed, but their use, recommended dosages, toxicity, and other factors are listed in several sources.[2,3]

TABLE II

ANTI-TUBERCULOUS AGENTS

FIRST LINE DRUGS	SECOND LINE DRUGS
Isoniazid	Ethionamide
Ethambutol	Pyrazinamide
Streptomycin	Capreomycin
Rifampin	Viomycin
Para-aminosalicylic acid	Kanamycin
	Cycloserine

The drugs in the right hand column are "second line" because they are almost never used as initial therapy in treating tuberuclosis. In addition, they have a high rate of toxicity and are less acceptable to the physician and to the patient. Second line drugs are frequently used in cases of re-treatment, treatment failure, or with resistant organisms (i.e. after first line therapy has proven to be inadequate or ineffective). Finally, these medications are often used in combination with some or all of the first line drugs in treating infections with a typical species of tuberculosis.

Drug Therapy: Treatment Regimens

As initial therapy in cases of active tuberculosis, at least two drugs should always be started. In instances of retreatment, the physician must know, through sensitivity testing, that the organisms in the sputum are sensitive to the drugs he prescribes.[2] In cases of suspected tuberculosis (where the PPD skin test is positive or the chest roentgenogram is abnormal, or both; but where the results of cultures are not yet known) two drugs are also started. If cultures are negative then only INH is continued for the remainder of one year. INH and ethambutol are presently used for initial treatment in a vast majority of patients.[10] This combination has the advantage of low toxitity, high effectiveness, ease of administration and low cost.[1] Many physicians add streptomycin or rifampin for the first 8-12 weeks in patients with extensive cavi-

tary disease. Experimental evidence suggests that such additional therapy can promote a more rapid conversion of the sputum to negative and prevent the emergence of resistant organisms.[6,7,8] It is now quite clear that the two-drug combination of INH and rifampin is also very effective in converting the sputum, although it is not used as often because of the relatively higher cost of rifampin, and because the INH-rifambin combination is additively toxic to the liver.[1,9]

Whenever additional drugs are required, two or more drugs are started; usually drugs with different mechanisms of action. In general, one of these is an injectable such as streptomycin.[1,2,6] The only exception to this rule is that a single drug such as rifampin, streptomycin, or pyrazinamide may be added if the patient is not responding rapidly to a two-drug regimen. This is done only if two-drug therapy has been in progress for less than one month and if the organisms are sensitive at low concentrations to the initial therapy. Failure to "respond" is frequently a function of other underlying problems such as alcoholism, malnutrition, or diabetes, rather than inadequate chemotherapy. Short courses of steroids have been advocated to augment the therapy in patients with persistent constitutional symptoms. Three drugs are always used in severe infections including: (1) advanced cavitary disease; (2) tuberculous meningitis; (3) miliary tuberculosis; (4) renal tuberculosis; (5) osseous involvement.

Intermittent Chemotherapy

Most treatment schedules call for daily dosages of pills and/or injections. Another approach to therapy, especially in unreliable or uncooperative patients, is the use of intermittent therapy under close supervision following discharge from the hospital. INH and streptomycin is one such effective combination. The INH is given in a dose of 14 mg/gk and streptomycin as 27 mg/kg usually administered twice weekly by the Visiting Nurse in the patient's home. Another such intermittent regimen combines INH and ethambutol administered twice weekly.[1,11] The use of drugs three times-a-week offers no advantage over twice-weekly programs.[1] In both the intermittent INH-streptomycin and IHN-ethambutol regimens, the results obtained are at least as good as with conventional daily programs.[1,11] Intermittent therapy is overall a more expensive approach, but the reduction in future reactivations undoubtedly will partially compensate for the additional cost. Because of severe, and potentially fatal toxicity, rifampin is not given to unreliable patients or used in intermittent therapy.

Short-Course Chemotherapy

Recently attention has been focused on the desirability of shortening the course of chemotherapy from the standard 18-24 months to six months. Several studies have reported favorable results from "short-course" chemotherapy.[12,13,14] One study conducted by the British Medical Research Council in East Africa[12] has followed several hundred patients; half treated with 3-drug therapy for six months and half treated with conventional 18-month chemotherapy. The study employed several drug regimens given for six months. Six-month courses of streptomycin-INH-thiacetazone and INH-streptomycin resulted in 22% and 27% relapse rates respectively at 1-year follow-up and were abandoned. However, when a 6-month course of streptomycin-INH-rifampin was compared to the 18-month control using streptomycin-INH-thiacetazone, the 6-month regimen and the standard 18-month regimen achieved very similar results at the end of a 5-year follow-up period. The bacteriologic failure rates during chemotherapy in patients with initially sensitive organisms were 0% (6-month) and 3% (18-month). The long-term bacteriologic relapse rate for the groups was 3% and 2% respectively. The majority of the relapses following short course therapy occurred within the first 12 months after the conclusion of treatment.

Of additional interest in this study was the fact that the authors came to regard pyrazinamide as a "sterilizing" agent useful in achieving a rapid bacteriologic conversion within the first two months of a six-month course. Pyrazinamide may eventually find its way into the "first line" drug category.[12]

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Drug Therapy Problems

Instruction For Patients On Oral Anticoagulation

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Lori Ulfers, R.N.
Tom Samuels, Ph.D.

INTRODUCTION

Over the past three decades, oral anticoagulation has been used to prevent the adverse effects of intravascular thrombus formation. Based on the premise that interference with hemostasis will reduce the mortality and morbidity associated with unwanted intravascular thrombus formation, prophylactic oral anticoagulation has become accepted medical practice.

It is beyond the scope of this paper to present a detailed review of the indications for oral anticoagulation, but a list of the most widely accepted indications and probable indications are presented in Tables I and II[1,20]

When the physician considers anticoagulation therapy, he weighs the theoretical benefit against the potential hazard and eliminates any patient with absolute contraindications to anticoagulation (Table III). In this equation, the reliability of the patient is a major consideration. Long term oral anticoagulation therapy should never be undertaken unless the patient is intellectually and emotionally willing to accept and share the responsibility for safe and effective anticoagulation with their physician.[21,22] Once this decision has been reached, adequate instruction of the patient is a time consuming problem. Shortcuts may have disastrous consequences. It is important that verbal instructions (Fig. 1) are enforced with written material to which the patient can refer to after the office visit (Fig. 2).

Existing patient instructional material appeared to us to lack the following: 1) appropriate emphasis on patient responsibility, 2) information about both of the widely used coumadin derivatices, 3) instruction on diet and foods containing high concentrations of vitamin K and, 4) information about prescription and non-prescription drugs that interact to potentiate or reduce the anticoagulant effects on warfarin. Accordingly, we developed the patient instruction manual that is presented in the Addendum. Physicians in Arizona will be supplied with these brochures upon request.



Figure 1

AN APPROACH TO ORAL ANTICOAGULATION

The majority of patients that we are currently following in our Anticoagulation Clinic are controlled on 2-10 mg of warfarin a day. No patient requires a daily maintenance of more than 10 mg daily, but 3/56 or 5% of our patients require a maintenance of less than 2 mg per day.

It has been recommended that the prothrombin time (PT) be regulated at 1½ to 2½ times control PT in seconds.[23,24] We have found that a prothrombin time of 2½ times control is too high, resulting in excessive bruising and bleeding. We are presently regulating the warfarin dosage of our patients at 1.7 to 1.8 times control. Thus, with a control of 11.8 seconds, the PT of the patient should be 20-22 seconds. If the control is 12.8 seconds, a patient's PT of 21.5 to 23 seconds is

TABLE I

INDICATIONS FOR ORAL ANTICOAGULATION

Pulmonary Embolism*
Deep Vein Thrombosis*
Arterial Embolism in Patients With:
—mitral valve disease
—prosthetic heart valves
—suspected site for mitral thrombosis
—atrial fibrillation
Mitral Valve Disease with Atrial Fibrillation
Prosthetic Heart Valves
Transient Ischemic Attacks
Acute Myocardial Infarction Associated With:

.

**following initial treatment with Heparin*

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ideal. In actual practice, we consider a patient prothrombin time between 20 and 24 seconds in our laboratory to be satisfactory. The control and patient prothrombin time may vary considerably from laboratory to laboratory, depending upon the thromboplastin used. If the control prothrombin time is greater than 14 seconds, the thromboplastin used is too weak and the results are not reliable.

A loading dose of warfarin is not necessary.[25] Like most drugs, the blood level of coumadin will reach 90% of its eventual maintenance or steady-state blood level after five half lives. The plasma half-life of warfarin is 42 hours. Therefore, warfarin accumulation, if a dose of the drug is given every day, will take approximately (5 x 42 = 210 hours) eight to nine days. Appreciation of the pharmacokinetics of warfarin is essential to adequate anticoagulation therapy. We begin therapy with a daily or maintenance dose. Two milligrams (mg) per day is used in the patient who is elderly, postoperative, debilitated, or who has suspected liver disease or liver congestion. All other patients are begun on 4 or 5 mg daily. Our personal preference is to start with 4 mg daily, that is, two 2.0 mg tablets daily. Then, adjustments can be made in 1 mg daily increments. If the patient is begun on 5 mg daily and this is found to be excessive, the usual physician response is to alter the dose to 5 mg one day and 2½ mg the next day. This results in unnecessary fluctuation in the prothrombin time and makes control



Figure 2

TABLE II

POSSIBLE INDICATIONS FOR ORAL ANTICOAGULATION

Atrial Fibrillation Associated With:
- —tachycardia-bradycardia syndrome
- —congestive heart failure
- —advanced age
- —rheumatic heart disease
- —idiopathic hypertrophic subaortic stenosis
- —attempted cardioversion

Thromboembolic events on or from predisposing lesions within the arterial tree.

Myocardial Infarction Associated With:
- —shock and/or prolonged bed rest
- —associated atrial arrhythmias
- —advanced age and/or obesity
- —history of embolism or venous thrombosis

Congestive Cardiomyopathy

Prevention of venous thrombosis in high risk groups
- —fracture of lower limbs
- —hip surgery
- —surgery of pelvis or lower abdomen which requires extensive dissection
- —varicose vein surgery
- —obesity, surgery & bed rest
- —cancer surgery
- —prolonged bed rest or immobilization
- —congestive heart failure

TABLE III

CONTRAINDICATIONS TO ANTICOAGULATION

- —lack of patient cooperation
- —active ulcer disease of GI tract
- —pre-existing coagulation defect or bleeding abnormality
- —severe liver or kidney disease
- —bacterial endocarditis
- —open ulcerative wounds
- —malignant or severe accelerating hypertension
- —contemplated or recent surgery of eye, spinal cord or brain
- —recent cerebrovascular hemorrhage
- —dissecting or ulcerating aortic aneurysms
- —hemorrhagic pericardial effusions
- —pregnancy (relative)
- —alcoholism
- —occupations that carry a substantial hazard of injury
- —inadequate laboratory facilities
- —compulsive drug users

more difficult. The prothrombin time is initially followed every other day if the patient is hospitalized and every fourth day if they are anticoagulated on an ambulatory basis. The dose is not increased until the patient has been on daily therapy for eight days, but can be decreased at any time that it appears that the daily dose will result in an excessive effect. Once control is achieved, prothrombin times are followed at 4-6 week intervals. Using this approach, associated with verbal and written patient instruction (see Addendum), we have found oral anticoagulation to be safe and effective.[26, 27, 28] Significant gastrointestinal and urinary tract bleeding during well-controlled anticoagulation therapy may indicate occult lesion and is an indication for further diagnostic tests.[29]

Oral anticoagulation is not 100% effective. Systemic emboli occasionally occur even when the patient's prothrombin time is in the desired therapeutic range. Recent studies in patients with prosthetic cardiac valves indicate that anticoagulation with the combination of warfarin and a controlled amount of salicylates is more effective than anticoagulation with warfarin alone.[30]

Since this article was submitted, an excellent autopsy study has appeared which shows that the incidence of clinically significant systemic embolism is almost as great in patients with atrial fibrillation and ischemic heart disease (35%) as in those patients with atrial fibrillation and mitral valve disease (41%).[31] The Framingham Study demonstrated that, even when all patients with rheumatic heart disease were excluded, there is a 5.6 fold increase in the incidence of strokes in patients with atrial fibrillation when compared to patients with normal sinus rhythm.[32] These two studies suggest that atrial fibrillation in any patient with heart disease may be an indication for anticoagulation and should be added to Table II. Controlled studies to determine if oral warfarin anticoagulation decreases the mortality and morbidity of patients with atrial fibrillation are needed.

ADDENDUM

ORAL ANTICOAGULANTS: INSTRUCTIONS FOR PATIENTS

PATIENT INSTRUCTION

Coumadin and Panwarfin (oral anticoagulants) slow the clotting process and prevent blood clots from forming inside the blood vessels. Your doctor has explained why it is necessary for you to take Coumadin or Panwarfin.

IMPORTANCE OF LABORATORY TESTS

The effect of oral anticoagulants varies from person to person. It is therefore important to test the effect of this drug at intervals for, if the dose is too high, you may have excessive bruising or internal bleeding. The dose of Coumadin or Panwarfin must be adjusted by a blood test called the prothrombin time or "protime." The "protime" will need to be drawn at least weekly for the first month. Thereafter, if steady control is achieved, the "protime" may be checked less frequently, such as 4 to 6 week intervals.

It is *your obligation* to make sure you have appointments for the "protime."

COUMADIN OR PANWARFIN

Coumadin (crystalline sodium warfin) and Panwarfin (sodium warfin) are available in several concentrations. The concentration of warfarin (in milligrams) is imprinted on each tablet. The tablets are "scored" so they can easily be broken in half. The concentrations or "sizes" available are 2, 2½, 5, 7½, 10 or 24 milligrams (mgs.) and each is distinctly colored. Coumadin has an identification number on the back side. All concentrations of Panwarfin have the symbol on the back side.

BOTH BRANDS	COUMADIN	PANWARFIN
2 mgs Lavender	(Endo 170)	
2½ mgs Orange	(Endo 171)	
5 mgs Peach	(Endo 172)	
7½ mgs Yellow	(Endo 173)	
10 mgs White	(Endo 174)	
25 mgs Red	(Endo 175)	

You must be familiar with these dosage forms. You may, for example, be asked to take 6 mgs a day. You would then either take three of the 2 mgs (Lavender) tablets or one 5 mgs (Peach) and ½ of a 2 mg (Lavender) tablet.

OTHER MEDICATION

Many drugs interact with oral anticoagulants. Some will reverse the anticoagulant effect with the hazard that a blood clot will form too easily. On the other hand, other medicines will interact with Coumadin or Panwarfin to prevent the blood from clotting. This may result in the same problems that occur with excess drug intake, such as the hazard of easy bruising or internal bleeding. You should *not take any medicine or discontinue a medicine* unless directed to do so by your doctor.

The following is a list of frequently used drugs that interact with Warfarin:

NEVER USE: Acetylsalicylic Acid (salicylates or aspirin)
Phenylbutazone (butazolidin, azolid)
Oxyphenylbutazone (taudearil, oxalid)
Indomethacin (Indocin)

Your physician may have to alter Warfarin dose and check prothrombin time more frequently when any of the following drugs are begun and/or discontinued:

- Allopurinol (Zyloprim)
- Anabolic-Adrogenic Steroids (Testosterone)
- Antibiotics, broad spectrum (Tetracycline and Neomycin)
- Barbiturates (Phenobarbital, Tuinal, Seconal, Nembutal, Butisol, etc.)
- Chloramphenicol
- Choloxin
- Cholestyramine (Questran)
- Clofibrate (Atromid)
- Corticosteroids
- Disulfiram (Antibuse)
- Ethchlorvyol (Placidyl)
- Glutethimide (Doriden)
- Griseofulvin
- Haloperidol (Haldol)
- Mefenamic Acid (Ponstel)
- Meprobamate (Miltown)
- Methylphenidate (Ritalin)
- Oral Contraceptives
- Quinidine & Quinine
- Rafampin
- Sulfonamides, long-acting
- Vitamin K & E

DO NOT TAKE ASPIRIN OR SALICYLATES. Tylenol, Datril, or Prompt (acetaminophen) should be used for minor aches or pains in place of aspirin.

Because salicylates are in so many drugs, they are the major problem. The following is a list of drugs that contain aspirin (salicylates). These should not be used.

ACA Caps #4
ACD
Acetonyl
Aidant
Aidant with Dover's powder*
Alka Seltzer
Allylgesic*
Allylgesic with ergotamine*
Alprine*
Aluprin
Amytal with ASA*
Anacin
Anexsia with codeine
Anexsia-D*
Anodynos
Ansemco #2 & 8
Apamead*
APC
APC with codeine*
APC with Demerol
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Aspirin-secobarbital Supprettes No. 1, 2, 3, & 4*
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Bayer timed-release
Brogesic*
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Buffadyne
Buffadyne with barbiturates
Bufferin
Bufferin arthritis strength
Buffinol
Arthra-Zene caps
ASA
ASA compound
ASA compound with codeine*
Asalco tab and Asalco #2
Ascodeen-30*
Ascriptin
Ascriptin with codeine*
Aspergum
Asphac-G*
Aspirin (USP)
Aspirin aluminum
Aspirin children's
Aspirin compound with Dover's powder*
Darvon with ASA*
Darvon-N with ASA*
Darvon compound 32 & 65*
Dasikon
Dasin caps*
Dasin-CS*
Dasin ¼ strength*
Decagesic*
Derfule*
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Colene compound 65*
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Emprazil
Emprazil-C*
Epragen*
Equagesic*
Excedrin
Excedrin PM
Fiorinal*
Fiorinal with codeine #1, 2 & 3*
Fizrin
Formasal caps & tabs
4-Way cold tabs
Gelsodyne*
Histadyl & ASA compound
I-PAC*
Liquiprin
Marnal tab & cap
Measurin
Midol
Niprin*
Calurin
Cama Inlay
Capron
Causalin*
Cheracol caps
Cirin
Codasa tab*
Colrex
Colrex compound*
Congesprin
Cope
Coralsone modified*
Coricidin
Coricidin "D"
Coricidin Demilets
Coricidin Medilets
Convangesic rub only
PC-65*
Percobarb*
Percodan*
Percodan-Demi
Persistin
Phenaphen*
Phenaphen with codeine
Phencaset improved
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Phensal
Pireseal
Predisal*
Pyrroxate
Pyrroxate with codeine*
Rhinex
Robaxisal*
Sal-Fayne
Salibar Jr*
Salipral*
Stanback
Stero-Darvon with ASA*
St. Joseph
St. Joseph for Children
Supac
Synalgos*
Synalgos-DC*
Tetrex-APC with Bristamin*
Toloxidyne
Triaminicin
Vanquish
Zactrin*
Zactrin compound*

Unlisted products identified in 5 community pharmacies:
- BiAct cold tabs
- Defencin
- Haysma

Norgesic*
Novahistine with APC
Novrad with ASA*
Opacedrin
Opasal
Pabirin
PAC compound tab
PAC compound with codeine*
Palgesic*
Monacet
Quiet World
Saleto
Sine-Aid
Soltice cold tabs

*On prescription only

WHEN TO TAKE YOUR TABLET

We recommend taking the anticoagulant tablet the same time each day, usually between the hours of 8:00 and 9:00 a.m. The "protime" should be drawn about 24 hours after last Warfarin tablet. On the day the "protime" is drawn, the tablet is not taken until after blood is drawn.

INTRAMUSCULAR INJECTIONS

Intramuscular injections should not be administered to you when you are taking anticoagulants since this may cause bleeding deep in the muscles.

HELPFUL HINTS

Guard against cuts; wear shoes. When gardening or doing similar work, wear gloves. The use of electric shavers is advised.

Do not stop taking your Coumadin unless directed by your doctor or nurse. Be sure you know how to obtain tablets before you run out. If you plan to be out of town for a period of time, be sure to discuss this with your doctor.

Women should notify their doctors as soon as they suspect they are pregnant.

Have your prescriptions filled at a pharmacy that maintains a patient drug profile. This way your pharmacist can identify possible drug interactions.

Avoid food fads, crash diets, or marked changes in eating habits. Certain foods are high in vitamin K. These need not be avoided; however, a marked change in the amount of these will alter your response to oral anticoagulation. These are alfalfa, cabbage, cauliflower, liver, soybeans, spinach, fish and fish oils.

The telephone number of the Anticoagulation Clinic at the Arizona Health Sciences Center is 882-7248.*

**You may want to have your own office name and phone number here.*

NOTIFY YOUR DOCTOR OR THE ANTICOAGULATION CLINIC IF:

You have red or dark brown urine.
You have red or black bowel movements.
You have a severe headache or stomach pain.
You have excessive bleeding after cuts.
You have excessive or unusual bleeding from any place on your body.
You have dizziness, nausea or fever.
You have bruising (black & blue spots), pain and/or swelling resulting from a minor bump.
You develop a skin rash.
WOMEN: You have a heavy or unusual menstrual period or if you become pregnant.

REMEMBER:

You should wear or carry identification indicating that you are taking warfarin.

You should contact your doctor prior to dental extraction or elective surgery.

Aspirin, some cold remedies, vitamins and sleeping pills affect the action of Coumadin and Panwarfin. Alcohol consumption may alter your response to Coumadin and Panwarfin.

Ask your doctor, nurse or pharmacist about taking any medication whether it is a prescription drug or "over-the-counter" medicine. Remind them that you are taking Coumadin or Panwarfin.

Developed by
Section of Cardiology
Department of Internal Medicine
and
Pharmacy and Supply
Arizona Health Sciences Center

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Medical Audiovisual Services
Arizona Health Sciences Center
Tucson, Arizona

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Emotional Disorders

Both Feet Firmly Planted In Mid-Air

William B. McGrath, M.D.

The physician assumes his obligation to encourage and to supervise, to some extent, the physical fitness programs of his patients. But are we defaulting our duty to the "whole man" by ignoring his mental fitness?

Disciplined mental exercising would contribute more to survival and productive longevity than does heredity or diet or jogging. In our times an individual has more use for a healthy intelligence than for the ability to overtake and spear a wild boar.

Yet here we anticipate a kind of pained resistance. We hardly feel qualified to teach mental fitness, and this is a serious flaw in our medical education. Our adult patients would not want to feel patronized. They had rather take for granted their ways of thinking and their use of language. Just as the amateur poet or painter declines formal instruction, people rationalize that they might lose spontaneity or creativity or their individual style.

Despite relatively secure circumstances, most people struggle with anxieties and non-specific nervous tensions. They are bled by unseen vampires. We owe it to them to insist on their devoting, say, fifteen minutes two or three times a week to mental exercising.

From: 5055 N. 34th St., Phoenix, AZ 85018.

Consider what is most basic: The awareness of abstracting. Illuminated consciousness of this one mechanism would spare a vulnerable population worlds of needless anguish and confusion.

A patient says: "I dread going home from the hospital." A second patient says: "I do, too. But when I get there I am going to put away the summer clothes and straighten up the closets." Unlike the first patient, she has brought reality into her field of anticipatory vision, thinking of tasks which are manageable and familiar. One cannot rehearse how to react to a murky feeling of dread. One can always cope better with the known than with the unknown.

A man has been having an affair with a woman. Five times he verbalized his conflict: "I don't know whether I want to keep this relationship or not." Finally we ask the young lady's first name. She became a flesh and blood person. Until then, talking about "the relationship," he had been arguing with himself as if whether to embrace the wind.

Consciousness of abstracting: A person observes an event. In order to remember and recount it he tells himself what he is observing. Otherwise, without inner speech, he could not store it in memory. But in flattening the experience into words he necessarily omits a myriad of perspectives and particulars.

Now there is nothing inherently erroneous about abstracting. As a matter of fact this is the ability which sets us apart from other creatures. We can share our learning over limitless gaps of space and time.

What distorts the reality testing of many people is their failure to realize when and to what degree they are abstracting. They want to know how to improve their self esteem or whether they "are" good or bad, strong or weak, mature or immature. You know how very frustrating it is to listen to someone who talks in broad generalities.

A small example:

1. Meet Harvey Snurp.
2. He is a plumber (the false "is" of identity; not what he does).
3. Plumbers make good money.
4. The plumbers' union is a monopoly.
5. Yes; and men get higher pay than women.
6. That's human nature.

Only when we shake hands with (denote) Mr. Snurp are we in contact with reality. At each successive elevation we have jettisoned more and more of his existence, until we are no longer talking about him at all. And at the higher levels of abstraction or generalization, we are in the rarified atmosphere of pure nonsense.

This is why so many of our proverbs and everyday expressions employ metaphors or similes (a train of thought; if the shoe fits; a flag-waving patriot). Instinctively we seek a referent, something accessible to the senses and specific; something real.

Reality testing—sanity—requires practiced consciousness of abstracting. Any information conveyed by language is incomplete and somewhat misleading. When we are mindful of this we are not quite so quick to make judgements. This is philosophical. This is wise. (A person cannot *be* wise; cannot *have* wisdom. He can use (verb) knowledge intelligently).

There are several ways of exercising the mind to improve its fitness. One is to occupy the allotted time in keeping a journal or diary. The person can then check his writings from a semantic point of view. Does what he has written have any reference to reality? Is he abstracting, generalizing, projecting, either-or-ing? On request we can point out to him the fallacies he has overlooked.

A more advanced exercise is to read one or another of the truly classical poems, asking at each inch, "Why did the author choose this word or that phrase?" Try "Ode to a Grecian Urn" or "The Hound of Heaven."

Another worthwhile calisthenic is to search the imagination for original similes to replace such hackneyed expressions as: weak as a kitten, lifestyle, pretty as a picture, stubborn as a mule, mad as a wet hen. Habitual dependence on cliches gives evidence of intellectual softness and obesity, shortening the life of the mind.

Educated persons, not just professionals, have an imperative duty to apply and to point out examples of the plain principles of reasoning. On a person to person basis this is what is (or should be) meant by effective psychotherapy. Most psychiatric patients simply do not know what they are talking about. Their agitation is the roilling of their corrupt and unrealistic mental processes. Psychotherapy is a dialectic, a dialogue, a tactful search for intelligent questions for which there finally might be sensible answers.

Does all this call for wider application? While I am writing the news reports that whites are killing blacks in Africa and blacks are killing whites, and elsewhere Israeles and Arabs are poised for mutual annihilation!

I do not know what "is" a Jew in these insane, pre-literate abstractions, nor what "is" a black (uncapitalized, one notes). Until he enters his place of worship we cannot tell an Irish Catholic from a Protestant. Notions of race or religion are abstractions, man-made artifacts. There is no such thing as "Jewish blood." And in the evening of humanity we'll not be able to see or remember whose skin is darker than our own.

Seminars in Gastroenterology and Liver Disease

Colo-Colonic Fistula Following Disruption Of Colo-Sigmoidostomy:

Or "Always Keep Your Options Open"

Alfred E. Stillman, M.D.
Francisco G. Quiroga, M.D.
Gerald Giordano, M.D.

Editors:
Leon Rigberg, M.D.
Stephen Glouberman, M.D.
George Burdick, M.D.

Summary:

A patient underwent left hemicolectomy for adenocarcinoma of the colon. Several months later she developed symptoms and signs of colonic obstruction. Re-exploration revealed total disruption of the colo-sigmoid anastamosis with bridging of the gap by a conduit composed of structures including omentum, peritoneum, left kidney and small bowel with associated mesentery.

Patients who suffer a disrupted colon succumb unless bowel continuity is rapidly re-established. We present a patient who developed an unrecognized post-operative disruption of a colo-sigmoid anastomosis. She survived due to the rapid spontaneous formation of a fistulous tract between the separated ends of bowel that prevented significant peritoneal contamination.

From the Departments of Internal Medicine and Surgery, Tucson Medical Center and the Tucson Hospitals Medical Education Program. Reprint requests to: Alfred E. Stillman, M.D., 2122 N. Craycroft, Tucson, Arizona 85712.

Case report:

A 68-year-old woman presented with a history of increasing obstipation and weight loss without rectal bleeding. Complete physical examination was unremarkable. A barium enema revealed an annular constricting sigmoid lesion consistent with adenocarcinoma; sigmoidoscopic examination failed to visualize the lesion.

Following bowel preparation with enemas and antibiotics, a radical left hemicolectomy was performed with removal of the splenic flexure and an annular sigmoid carcinoma. Bowel continuity was re-established low in the pelvis and without tension by a 2-layer termino-terminal anastamosis between the sigmoid and transverse colon. The neoplasm had infiltrated to the serosa; two mesenteric nodes were involved with tumor but the liver was free of metastases. The patient tolerated surgery without incident. During the post-operative course, the patient

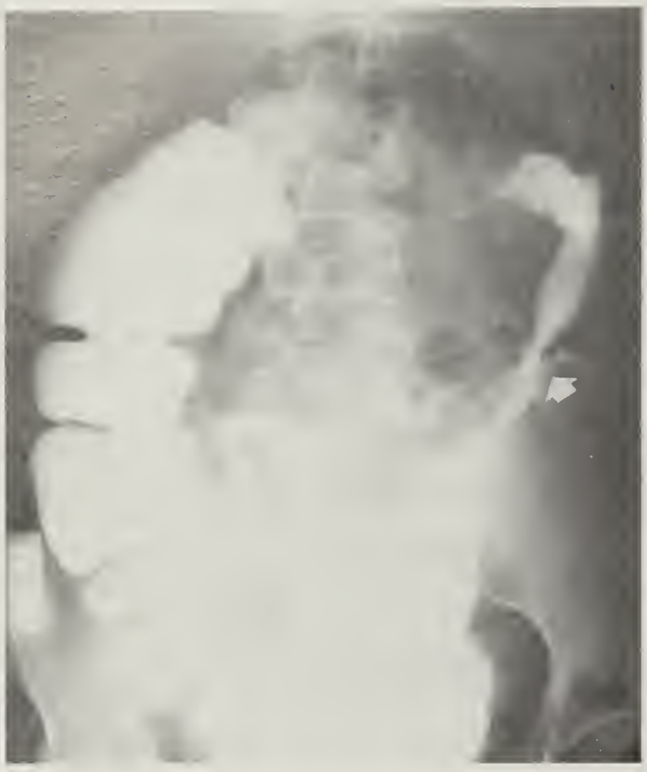


Figure 1
The x-ray demonstrates a stricture with sinus tract (arrow); there is no identifiable mucosa in the stenotic segment.

developed fever to 102° for 17 days. The source of the fever was never determined with certainty during that admission but gradually resolved after massive antibiotic therapy.

Two months following surgery, the patient again developed increasing obstipation associated with rectal bleeding. Physical examination revealed only mild left lumbar tenderness. However, a barium enema (Fig. 1) delineated a stricture 8 cm long at the anastamotic site. The stricture presented a nodular appearance with sinus tracts at the distal end; no normal mucosa was seen. Colonoscopic examination (Fig. 2) revealed a stenotic area at 20 cm from the anal verge surrounded by nodular, friable tissue; biopsies of this area obtained under direct vision demonstrated only normal colonic mucosa. It was not possible to pass the instrument beyond the distal end of the stricture.

The patient underwent exploratory laparotomy three months after the original colonic resection. A gap 8 cm long was discovered between the separated ends of the sigmoid and transverse colons. This gap was bridged by a conduit whose walls were composed of greater omentum, posterior peritoneum, left kidney and small bowel with associated mesentery. No recurrent carcinoma was found. The adherent omentum, posterolateral peritoneum and left kidney were dissected free and adhesions binding loops of small bowel to the fistulous area were lysed. End-to-end colosigmoidostomy was then performed. The patient had a stormy post-operative course but left the hospital in good condition.



Figure 2
Colonoscopic view of the distal end of the stricture revealing nodularity consistent with carcinoma.

Discussion:

This unique patient dramatically illustrates the remarkable ability of the body to spontaneously localize intra-abdominal infection. The anastamotic disruption undoubtedly occured early in the post-operative course and was manifest only by fever. Fortunately, early and vigorous antibiotic therapy eventually eradicated the intra-abdominal septic foci resulting from the initial leak. When the patient presented with obstructive symptoms three months following the first operation, the radio-logic and colonoscopic appearance favored a diagnosis of carcinoma but three months seemed too short a time to allow so extensive a recurrence. Moreover, colonoscopic biopsies were unable to prove the presence of neoplasm. The pre-operative diagnosis was stricture secondary to bowel ischemia, an uncommon complication of left hemicolectomy that may occur consequent to ligation of the inferior mesenteric artery.[1]

One previously reported patient developed necrosis and autoamputation of a sigmoid volvulus; stool flow was maintained between the disrupted colon ends through an abscess cavity.[2] Our patient is unique in that stool flow was preserved through an anatomically integral tract that prevented fecal contamination of surrounding structures. A recent comprehensive review of intestinal tract fistulae did not mention a colo-colonic fistula similar to that found in our patient.[3] In retrospect, the historical, radiologic and colonoscopic observations in our patient were entirely consistent with the surgical findings at the final operation.

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Final classification of the less-than-effective indications requires further investigation.

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Intramuscular: 5 to 10 mg. (1 or 2 ml.) two or three times daily. Intramuscular administration may be used initially in severe or acute conditions.

Contraindications and Cautions: There are no known contraindications to oral use when administered in recommended doses. Should not be given immediately postpartum or in the presence of arterial bleeding.

Parenteral administration is not recommended in the presence of hypotension or tachycardia.

Intravenous administration should not be given because of increased likelihood of side effects.

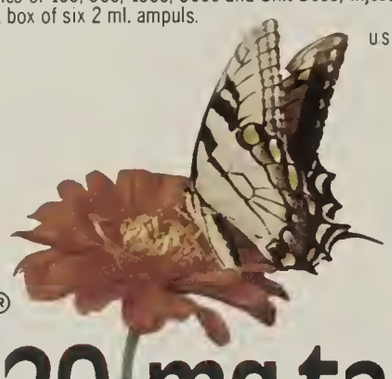
Adverse Reactions: On rare occasions oral administration of the drug has been associated in time with the occurrence of hypotension, tachycardia, nausea, vomiting, dizziness, abdominal distress, and severe rash. If rash appears the drug should be discontinued.

Although available evidence suggests a temporal association of these reactions with isoxsuprine, a causal relationship can be neither confirmed nor refuted.

Administration of single dose of 10 mg. intramuscularly may result in hypotension and tachycardia. These symptoms are more pronounced in higher doses. For these reasons single intramuscular doses exceeding 10 mg. are not recommended. Repeated administration of 5 to 10 mg. intramuscularly at suitable intervals may be employed.

Supplied: Tablets, 10 mg., bottles of 100, 1000, 5000 and Unit Dose; Tablets, 20 mg., bottles of 100, 500, 1000, 5000 and Unit Dose; Injection, 10 mg. per 2 ml. ampul, box of six 2 ml. ampuls.

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Precautions: Do not administer more frequently than every 6 hours, or within 12 hours after rectal dose of any preparation containing theophylline or aminophylline. Do not give other xanthine derivatives concurrently. Use in case of pregnancy only when clearly needed.

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How Supplied: Capsules in bottles of 100 and 1000 and unit-dose packs of 100; Elixir in bottles of 1 pint and 1 gallon.

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Obstetrics and Gynecology

Drugs In Pregnancy Part I

David Reid, M.D.

Donald J. Ziehm, M.D. Editor

Any drug administered to the pregnant woman may have a detrimental effect on the fetus. Scores of new drugs have appeared during the last 15 years. With their introduction physicians have become acutely aware of the potential for trouble. The Thalidomide tragedy remains a perpetual warning. Vigilant surveillance of the medications patients are using, whether prescribed or over-the-counter, is essential.

One patient's meat is another patient's poison, especially when they are linked by the placental circulation. What the mother consumes the fetus will get, like it or not. Easy transport across the placenta is available for drugs of low molecular weight. The quantity of medications used by pregnant patients is incredible, as demonstrated repeatedly in the literature. Prescription drugs, over-the-counter preparations, social drugs, food additives, environmental pollutants, etc.—anything to which an individual can be exposed should be suspect. Unfortunately, most physicians do not elicit this type of information when taking a history.

Presented at Phoenix OB-BYN Society 10/20/77.

Dr. Reid, Clin Prof. OB-GYN, Univ. of Alberta.

The fetus may be affected in two ways by the medications its mother ingests. The direct effect, teratogenesis, is the most worriesome. In addition, deviations occur in fetal pharmocology which could ultimately produce neonatal problems and learning disabilities. The indirect effects concern: 1) placental dynamics; 2) uterine activity; 3) alterations in maternal metabolism with subsequent sequelae in the fetus; 4) the maternal cardiovascular system, possibly affecting the placental vascular bed; 5) the maternal respiratory system with potential modifications in maternal and fetal oxygenation.

Ensuing teratogenic effects are determined by the phase of organogenesis when the fetus is exposed to a chemical insult. *When* a drug is ingested, and its *dose level* will regulate what malformation or defect may develop. The teratogentic dose of an agent is between the amount which does not cause permanent fetal damage and that which may cause fetal death. Genetic predisposition can modify the effects of exogenous influences so that teratogenicity may vary between individuals, strains and species. Each fetus has an individual susceptibility to a particular drug. Thus, every woman taking Thalidomide did not have a

deformed fetus. Individual variation and genetic factors can control each woman's metabolic rate and method of metabolizing a drug.

The same chemical may cause various types of malformations, but different agents may result in similar anomalies. For example, many drugs may produce CNS malformations, but any particular compound may cause dissimilar abnormalities at different gestational ages.

Teratogenic effects to the fetus can happen at various times: CNS—15-25 days; heart and limb—20-40 days; limbs alone—24-46 days. The majority of potentially lethal or damaging defects occur before the 56th day of gestation, dating from the onset of menses. Therefore, most have developed almost before the second missed period. Before pregnancy is confirmed, a large number of medications may already have been used. This, of course, is disturbing.

The genital structures, CNS and other organ systems are also vulnerable at later stages of development. Thus, if a mother is taking Dicumerol or Thiazides, the fetus may develop thrombocytopenia or a bleeding diasthesis. However, the drug effect may be evident only later in post-natal life or in adulthood (Tetracycline, DES, etc.). Therefore, drugs administered to a pregnant woman may affect her child any time throughout its entire life span, an important fact to consider when prescribing medications during pregnancy.

Drugs may adversely affect the mother, fetus, or neonate. The altered physiology of the pregnant woman may modify the rate of metabolism, transfer and excretion of any compound. Obviously it is not possible to study potentially dangerous drugs in pregnant humans. Consequently animal studies are relied upon and, unfortunately, these are frequently not transposable to the human. Also, the placenta is capable of transferring both exogenous and endogenous substances across its cellular layers. Drug transfer may be influenced by any metabolic and/or functional deviation in the placenta. Placental aging and disease processes often influence rates of drug diffusion and transfer across its membrane. Certain maternal conditions (diabetes, toxemia, etc.) frequently result in altered drug transport due to morphological alterations in the placenta. As a general rule, drugs present in sufficient concentration on the maternal side of the placenta will eventually reach the fetus. The amount may vary early or late in placental development, but almost all medications will reach the fetus. Decreased placental circulation secondary to a detrimental influence of a drug in turn can influence fetal nutrition and oxygenation. In addition, drugs concentrations can vary in the fetus, amniotic fluid and maternal blood.

Where does the fetus fit into this whole picture? The amount of drug affecting fetal tissues is dependent upon the equilibrium between drug concentration in the maternal and fetal blood. The distribution in the fetus is variable. The ultimate effect of a compound is determined by the fetus's ability to metabolize or inactivate a drug for excretion. The immaturity of enzyme systems causes drug metabolism to vary markedly. As a result the fetus is susceptible to concentrations of drug far from lethal in the mother, which may cause severe problems in the fetus. The CNS, very vulnerable during periods of myelinization, is of great concern. Incomplete myelinization could theoretically be associated with impaired intellectual development, the so-called "learning disabilities." Might some of these drugs (tranquilizers and other therapeutic agents) cause long-term learning, developmental or behavioral impairment?

The treatment of possible disease in the fetus may necessitate administering large amounts of drug to the mother in order to produce a specific result in the fetus. In this instance the drug is given to the mother, thereby attempting to produce therapeutic concentrations in the fetus. For example, large doses of Penicillin are given to cure Syphilis in the mother and to prevent involvement in her fetus.

The infant is frequently exposed to drugs immediately before delivery, sometimes for the treatment of pre-existing disease or pregnancy-related disorders, but more commonly for pain relief during labor. The fetal problem?—how does he excrete or metabolize these medications. Once the umbilical cord is severed the baby is without the support of the maternal circulation, and suddenly no means are available for disposing of metabolites or drugs present at birth. If the mechanisms for drug metabolism are not well developed the newborn may excrete medications very slowly. Therefore the neonate, with its immature enzyme systems, may require longer periods before drugs can be adequately metabolized. Days may be necessary for the neonate to excrete a drug that an adult can handle in minutes or hours. The majority of drugs, whether unchanged or metabolized, are primarily excreted via the kidneys. Because the fetal kidneys are usually not fully developed, decreased ability to eliminate drugs may result.

In addition to drugs, substances present in the environment may have an impact on the embryo and germ cells. Radiation, chemicals, dietary insults, infection, hypoxia, temperature, endocrine imbalances, visible trauma, placental failures—all may harm the developing fetus. Several mechanisms may be operating within the embryo to give rise to difficulties: 1) chromosomal mutations—bothersome because of the unknown effect in the offspring of the fetus; 2) lack of precursor or substrates; 3) altered energy sources—ATP or high energy phosphates; 4) change in membrane characteristics; 5) water and electrolyte imbalance.

To Be Continued

Bioassay Of Human Tumor Stem Cells

A New Approach To Evaluation And Treatment Of Cancer

Sydney E. Salmon, M.D.

Editors:
Stephen E. Jones, M.D.
Robert H. Thoeny, M.D.

Cancer chemotherapy has improved considerably in recent years. However, the selection of effective chemotherapy for individual patients remains somewhat of a trial and error procedure, since predictive techniques (similar to the culture and sensitivity assays used for bacterial infections) have not been available. Many clinical observations have indicated that there is a wide range of responsiveness to particular treatments even among patients with cancers of the same histopathology. These differences in responsiveness have been observed even in cancer patients who are well matched with respect to tumor burden (stage) and other known prognostic factors. Thus, differences in responsiveness must be due to either differences in drug metabolism or inherent differences in tumor cell sensitivity to particular drugs.

Tumor stem cells are the cell renewal source of a neoplasm and also serve as the seeds of metastatic spread of cancer. Studies of transplantable tumors in animals indicate that tumor stem cell colony-forming assays (*in vivo* or *in vitro*) can be used to study the biological properties of these cells and to delineate differences in individual sensitivity to a variety of anti-cancer drugs.[1,2] For example, the development of an *in vitro* colony-forming assay for stem cells from transplantable mouse myeloma[3] (a plasma cell neoplasm) permitted detailed analysis of the effects of anti-cancer drugs *in vitro*, and the assays are predictive of response to treatment even in animals with advanced mouse myeloma.[1]

The ability to grow colonies from primary tumor cell explants in semisolid culture media has even greater potential application. Unfortunately, primary explantation of human tumors for colony formation *in vitro* had met with little success until a new approach to simple bioassay was developed by cell biologist, Dr. Anne Hamburger, and myself.[4,5] The technique uses an enriched tissue culture medium plus growth stimuli in a "feeder layer" and permits human tumor stem cells to grow and form colonies in soft agar. As is summarized in Table I, we have been successful in obtaining human tumor colony growth directly from biopsy samples from both solid tumors and hematologic malignancies. Thus far, we have performed bioassays for tumor colony formation in over 250 patients with cancer. The tumor samples are obtained by biopsy and kept under sterile conditions while a single cell suspension is prepared for culture. In the final preparative step, the tumor cells are mixed directly with molten agar and plated. In the soft agar petri dish cultures, tumor colonies grow in a fashion analogous to bacterial colonies and can be visualized and counted through an inverted microscope. A

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TABLE I

Types of Human Cancer From Which Successful Tumor Colony Formation Has Been Obtained*

A. Hematologic Malignancies	B. Solid Tumors
Multiple Myeloma	Adenocarcinoma of the Ovary
Non-Hodgkin's Lymphoma	Adenocarcinoma of the Endometrium
Hodgkin's Disease	Melanoma
Chronic Lymphocytic Leukemia	Undifferentiated Carcinoma (head & neck)
Waldenstrom's Macroglobulinemia	Small Cell Carcinoma (lung)
	Neuroblastoma

Not all tumor categories have yet been tested. To date we have had limited experience with gastrointestinal or breast carcinoma.

typical tumor colony appears in Figure 1. Specific histochemical and immunofluorescent stains have been utilized to prove that the tumor colonies consist of cells of the same type as those present in the biopsy sample. Dr. John Davis in our Pathology Department has been quite helpful in reviewing slides prepared from the tumor colonies and comparing them with the original biopsies. The plating efficiency is low (in the range of 1.0% or less). This suggests to us that either the number of tumor stem cells is quite small (which is quite likely), or that the nutritional conditions of culture can still be improved. In general, the tumor colonies can be counted 2-3 weeks after plating.

Thus far, we have focused on metastatic tumors which are readily accessible for repeated biopsies, and on clinical circumstances wherein we will have opportunity to make correlations between the results of tumor colony studies and the patient's clinical course and response to treatment. The technique is still a research procedure, and is being carried out with support from a grant recently awarded to the AHSC Cancer Center by the National Cancer Institute. To date, our highest success rates have been in ovarian carcinoma, metastatic melonoma, and multiple myeloma, wherein tumor colony formation occurs in over 75% of tumor samples plated. We have also had success with the various lymphomas. Clinical oncologists at AHSC have been responsible for the tumor biopsies, patient followup and clinical correlations for the various patient categories. Drs. David Alberts and Jack Schmidt coordinate the gynecologic cancer studies, while Drs. Brian Durie and Stephen Jones are responsible for myelomas and lymphomas, respectively. Various Arizona physicians have referred patients with melanomas and other forms of accessible cancer for these research studies. The excellent cooperation and referrals from throughout the state have been invaluable for these studies.

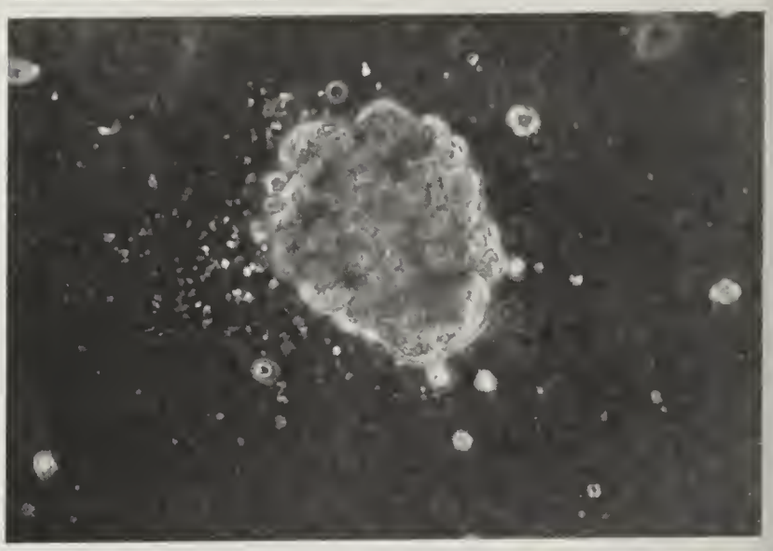


Figure 1
Typical melanoma colony in soft agar 11 days after plating (200X). Cytoplasm of the melanoma cells in the colony is stippled with melanin granules. In this case, colonies were grown from a solitary cerebral metastasis resected from a 27-year-old male with metastatic melanoma.

DRUG SENSITIVITY

Once the basic technique for bioassay was well established, we turned our attention to measurement of *in vitro* drug sensitivity. We have recently observed that quantitative measurements of tumor stem cell sensitivity to anti-cancer drugs can be obtained with this system. Prior to plating, the tumor cells are exposed to various concentrations of clinically useful anti-cancer drugs (e.g., melphalan, adriamycin, BCNU, bleomycin, vinblastine, DTIC) for 1 hour, after which the cells are washed free of the drug and cultures are prepared. Drug sensitivity is measured as the percent inhibition of tumor colony formation at various dose levels. Of interest, individual patient's tumors manifest unique *in vitro* sensitivity patterns (e.g., sensitivity to melphalan, resistance to adriamycin, resistance to bleomycin, sensitivity to vinblastine) in a fashion similar to that observed with various bacterial strains to antibiotics. Thus, inhibitory concentrations can be determined, and we can calculate whether such drug levels can be attained clinically based on the pharmacology of the specific drug. At the present stage we have been focusing our major efforts making clinical correlations in previously untreated patients with disseminated cancer between the *in vitro* results and the *in vivo* effects of treatment (as opposed to selecting treatment on the basis of the bioassay). In a series of more than 25 patients with myeloma, ovarian cancer, or melanoma we have observed both sensitivity and resistance to various drugs *in vitro*. The *in vitro* results thus far appear to correlate relatively well with *in vivo* sensitivity or resistance to the same agents.

Because of the encouraging findings in these correlative studies, we have begun to perform *predictive* culture and sensitivity tests for patients in

TABLE II

Cancer Patient Referral Categories Currently Requested for Investigative Study Of In Vitro Drug Sensitivity

Ovarian Carcinoma
Accessible tumor or malignant effusion

Multiple Myeloma
Untreated or in relapse

Non-Hodgkin's Lymphoma
Accessible lymph nodes or bone marrow involvement

Metastatic Melonoma
Accessible metastases

relapse. In two patients with ovarian carcinoma in relapse from standard chemotherapy, a specific treatment was selected on the basis of *in vitro* sensitivity. In both instances, clinically useful tumor regression occurred with the specific drug selected. We obviously are quite interested in pursuing this promising area of investigation by expanding our clinical research in the area of predictive drug and radiation sensitivity testing. Looking into the future, and assuming continued success with our approach, we would predict that the optimal setting for such bioassay studies would be at the time of primary surgery. Such tumor stem cell bioassays would appear reasonable for patient categories wherein adjuvant chemotherapy is considered because the tumor type is known to have a high likelihood of occult micrometastases. In that circumstance, bioassay results could be available shortly after wound healing is complete, and an individualized adjuvant chemotherapy program could be planned on the basis of measured drug sensitivity. As mentioned above, close cooperation and referral of suitable patients for our investigations by Arizona's physicians have contributed substantially to our success to date. Patient categories that would be particularly helpful at the present stage in our predictive testing studies are summarized in Table II. As we perfect the techniques for routinely culturing other forms of human cancer, we will of course extend our *in vitro* sensitivity testing program to those tumors as well.

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Ophthalmology

Macular Degeneration

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Editor:
Robert Rubenzik, M.D.

SUMMARY

The fundus disorder usually named senile macular degeneration is discussed here for the purpose of alerting the primary physician to characteristics which make early diagnosis possible. A description of the disorder in its several stages and forms is offered and a summary of the pathophysiological alterations is given. Available treatment is detailed.[1]

BACKGROUND

The term degeneration is defined in Dorland's Medical Dictionary as "deterioration; change from higher to lower form; especially change of tissue to a lower or less functionally active form."[1,2]

The macula (more properly the macula lutea) is an avascular area in the center of the posterior fundus of the eye, approximately the same size or somewhat larger than the optic nerve head.

While the term senile macular degeneration functionally fits the description of "deterioration," the etiology of this group of disorders remains obscure. There are indications that, in the future, this group may be described as an abiatrophy. The incidence of loss of macular function with characteristic cytoarchitectural change is unknown at this time. Predilection for sex or race seems unlikely. Genetic patterns, if present, are yet to be defined. Association with systemic disorders such as arteriosclerotic cardiovascular disease, hypertensive cardiovascular disease and collagen vascular disease is suspected, but remains unconfirmed at this time.

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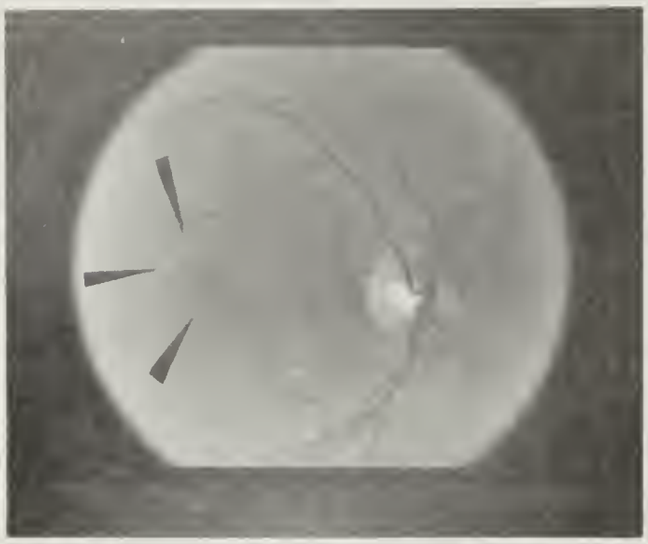


Fig. 1A
Early drusen in an asymptomatic patient.



Fig. 1B
Asymptomatic patient with drusen and arteriosclerotic "hard exudates" coexisting.



Fig. 1C
Many more drusen apparent with fluorescein dye (same patient as Fig. 1A)



Fig. 1D
Normal fundus for comparison.



Fig. 2
Early Stage III lesion with subretinal hemorrhage and scarring.

Stages of Macular Degeneration:
Dry — Wet
I. Drusen
II. Retinal Pigment Epithelium Atrophy & Subretinal Neovascularization.
III. Hemorrhagic Retinal Detachment & Scarring.

Fig. 3



Fig. 4A
Fundoscopic appearance of patient with early subretinal neovascularization and localized serous retinal detachment.

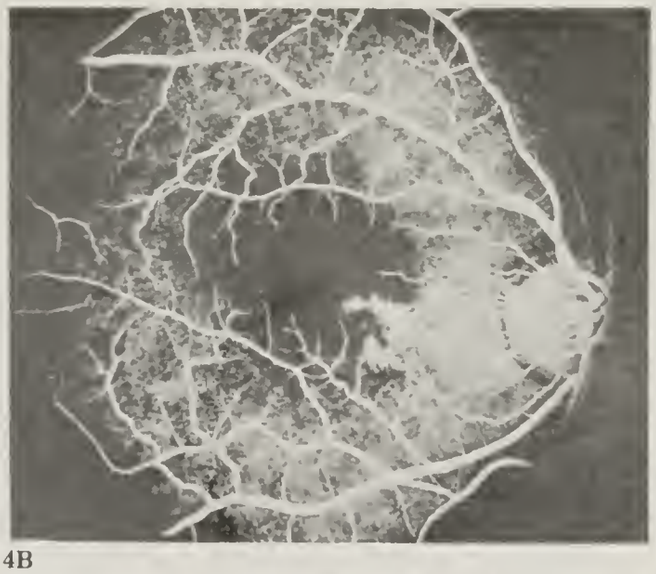


Fig. 4B
Fluorescein angiographic documentation of a subretinal neovascular lesion.

DISCUSSION

If recognition of early macular degeneration must depend upon the patient's awareness of symptoms, in most cases the disorder will be found in the late stages and may be too far advanced for treatment. Early symptoms if confined to one eye may escape the recognition of the patient. When symptoms are reported, they often take the form of slight distortion or mild decreased visual acuity. In most cases, however, it will be the physical signs noted by the patient's primary physician which will allow the most effective early diagnosis and treatment.

During a routine general examination, the most accessible portion of the fundus is the macula or central area of the posterior segment of the eye. An ophthalmoscopic examination of the macula may be the least taxing of all steps in the examination of the fundus. The ophthalmoscope should be routinely employed for this purpose. Senile macular degeneration may be identified prior to the onset of any symptom by its topographical fundoscopic hallmark —drusen. These small, round, whitish-yellow spots, previously thought to be benign excrescences of Bruch's membrane, are now believed to be associated with changes in the retinal pigment epithelium and choriocapillaris. Figures 1A and 1B show the appearance of a fundus containing drusen in an asymptomatic patient. In 1B arteriosclerotic hard exudates are included for comparison. Figure 1C demonstrates the appearance of the fundus from the patient in 1A on fluorescein angiography. This is a frequently performed, simple and objective diagnostic test used by ophthalmologists to identify and classify this disorder. It is especially striking to notice the number of drusen which appear in the presence of fluorescein dye when the appearance on plain ophthalmoscopy may be relatively benign. Figure 1D shows a normal fundus for comparison.

The finding of drusen in a fundus is indication for referral to the ophthalmologist. It is necessary to specify that drusen are not pathognomonic for macular degeneration, nor do they always precede further changes. Their incidence is unknown. In general, this degeneration of choroidal and retinal tissue is divided into two groups. The first group is a "dry" form with atrophic changes only. There is loss of the choriocapillaris and the retinal pigment epithelium in a disciform area occupying part or all of the macula. In the second form, a "wet" transformation is noted. An insidiously progressive subretinal fibrovascular membrane infiltrates the macular region.[3] This membrane, containing abnormal new blood vessels, weeps serous fluid and occasionally undergoes hemorrhagic change. Progressive edema of the overlying retina may be related to incompetent exchange of metabolites and causes distoriton and limitation of acuity. The secreted product of this membrane eventually causes a local serous detachment of the neurosensory retina. Hemorrhagic detachment is not unusual (Fig. 2). Chronic persistence of these changes leads to various degrees of gliosis or scarring of the lesion.

While the appearance of the macula may vary considerably from patient to patient, and during the course of the disease in an individual patient, the results of progression are a loss of central vision. In addition to the separation between "dry" and "wet" changes a staging method to simplify management is used (Fig. 3). In the earliest stages, when only a small number of drusen are seen, the patient is described as Stage I (Fig. 1A). When macular edema, subretinal neovascularization, or early focal hemorrhage are noted, the patient is given the designation of Stage II (Fig. 4A). During the evolution of the disorder, a Stage I or Stage II lesion may exhibit spontaneous arrest, without further progression. In Stage III, the macula may show either a dry atrophic lesion (Fig. 5), with drop-out of choriocapillaris and retinal pigment epithelium or an advanced neovascular mound with



Fig. 5
Stage III atrophic scar (dry).



Fig. 6
Stage III - white mound.

hemorrhage and scarring. End-stage lesions may present with an elevated white mass in the macula (Fig. 6). Disorganization of fundus topography may also take place (Fig. 7).

The natural history of macular degeneration is one of progressive distortion in the central visual field, followed by loss of central visual acuity. Remissions occur occasionally. Initially, distortion may be accompanied by 20/20 to 20/30 visual acuity. End-stage lesions often exhibit 20/200 vision, or poorer. The incidence of spontaneous resolution of an active lesion is thought to be small. However, there is an occasional patient in whom a lesion, eccentric to the fovea, may undergo limited regression. In such rare cases, central acuity may be partially spared. Since the disorder is limited to the macula, ophthalmologists reassure patients that they will not be blind as a result of this process.

For many years, the disciform macular lesion was considered to be secondary to occlusion of a vessel, but subsequent studies by Hayreh and others have demonstrated that there is no single artery responsible for macular supply. Rather, it seems that subdivisions of the short, posterior ciliary arteries supply the choroid and retinal pigment epithelium in a segmental distribution. Hayreh has stated that once a posterior ciliary artery is occluded, causing a filling defect in the fundus, the defect does not resolve. The lesion does not extend, but retains its borders. This statement would be inconsistent with the smouldering, progressive course of senile macular degeneration.[4]

The most disastrous component of the degenerative lesion has been identified as a subretinal neovascular membrane. To effect remission, therapeutic measures must prevent formation of this portion of the lesion, or selectively destroy it. Many medical regimens have been proposed but none have been generally accepted as effective. In 1974, a new treatment was proposed by Hruby, in Austria, utilizing an extract of pork retina called Etaretin.[5] The Boswell Hospital Proceedings reported experience with intravenous histamine for the same purpose.[6,8] Vitamins A and E have been utilized. Dietary alterations have been proposed. Peripheral vasodilators, anti-coagulants and anti-inflammatory drugs have been evaluated. Although proponents of various regimens appear enthusiastic, no controlled research program, to date, has demonstrated arrest or reversal of the disease process.

Furthermore, proponents of various medical treatments for macular degeneration have, to my knowledge been unable to show any significant change in appearance of the fundus after use of their treatments.

Various diagnostic modalities are in common usage by ophthalmologists. The most objective of these are fundus photography and fundus fluorescein angiography (as previously mentioned). These studies graphically show the vascular condition of the choroid and the retina in the given subject. (Figs. 4A & 4B).

Visual acuity is always recorded, but variability in the subjective acuity test precludes objective conclusions in this disease.

Other modalities which have been proposed to measure macular functions include visual field testing and color vision testing. Visual fields indicate areas of disturbed visual function and may improve and regress periodically with recurrent serous fluid formation, hemorrhage, and recurring edema in the retina. This is also a subjective test. Color vision is an inappropriate test since the cone apparatus, which is responsible for color perception, extends usefully to the periphery of the retina. Therefore, even a patient with a macular ablation should be able to report relatively normal color perception utilizing uninvolved peripheral cones. The reason that color perception, as presently tested, varies among macular degeneration patients is that visual acuity may be so reduced in the central field that the patient may not see the color vision target presented to him, using standard tests such as the ISHIHARA plates. This makes color vision testing inaccurate unless special effects are



Fig. 7
Disorganized macula.



Fig. 8
Postoperative appearance of macula with photocoagulation scars. Retina is now flat.

used, only because of poor central acuity.

It is to be expected that appropriate electrophysiologic testing will soon be available to objectively assess macular function. The focal electroretinogram and the focal visually evoked cortical response tests are currently under investigation in various centers for this purpose.

Laser and Xenon are photocoagulation techniques currently in use as acceptable means of controlling the proliferation of subretinal neovascular membranes in this disease. Success has been reported in cases selected for the location and size of the lesion. Currently photocoagulation of this type is the accepted therapy. These are destructive modalities and require skill in evaluating good quality fluorescein angiograms prior to treatment. Experience and attention to detail are mandatory in the use of these instruments. Of the two techniques, the laser is preferred because of its great versatility and accuracy. (Fig. 8). In avoiding the fovea, spot sizes as small as 50 microns may be applied using models presently available. Lesions are made surrounding areas of subretinal neovascularization, selectively destroying them. This modality is inappropriate for dry lesions or end-stage scars. It is not used when the fovea is directly involved or when the lesion is too extensive to control.

The greatest tragedy of this disease is an indifference sometimes exhibited when we are confronted by patients with advance senile macular lesions. Physicians tend to regard these problems as intractable. Actually, with special attention to training in the use of low vision aids, some of these people can learn to resume reading. They can play cards, work at hobbies, and engage in other limited manual tasks. They can find their way about and care for their personal needs. I have known of cases where related visual problems, such as cataracts, have been ignored because of the poor visual prognosis in this disease. It should be remembered that a patient with a loss of central acuity and 20/200 vision is far better able to function than a patient with only "hand motion" acuity secondary to cataract.

In the combination of lenticular opacification and macular degeneration, the intraocular lens implant finds great utility. Conventional cataract surgery leaves a patient with crippled peripheral vision because of spectacle distortion. Contact lenses are difficult to manage because of poor central visual acuity. Implants restore a relatively wide, useful, peripheral visual field. The patient with macular degeneration of the "senile" type deserves our attention early in his disease. In late stages, he is desperate for rehabilitation, he should be provided with both attention and rehabilitation whenever possible.

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AM Radiology

Case Of The Month # 28

Jonathan M. Levy, M.D.
Cary J. Stegman, M.D.

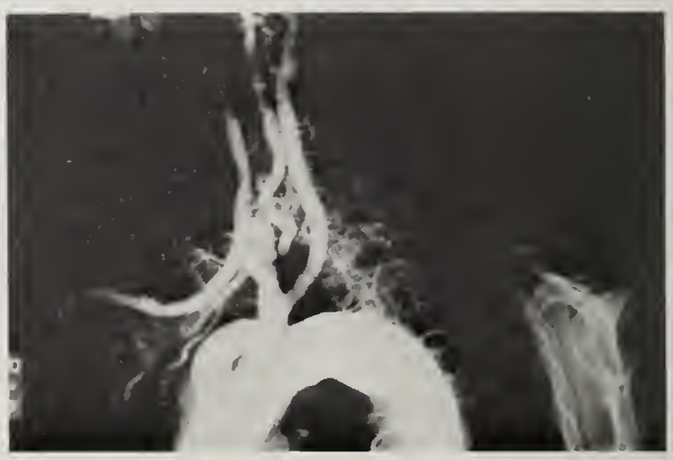


Figure 1

A 64-year-old man presented to his physician with pain in his left arm, which became worse whenever he worked in his garden. Because of these symptoms, as well as dizziness and a bruit in his neck, he was referred for arch aortography. A representative film from the arch aortogram is shown in Figure 1.

What are the major branches of the aortic arch?

Is any branch missing on the patient's aortogram?

How does this relate to the patient's symptoms?

Answer on page 119.

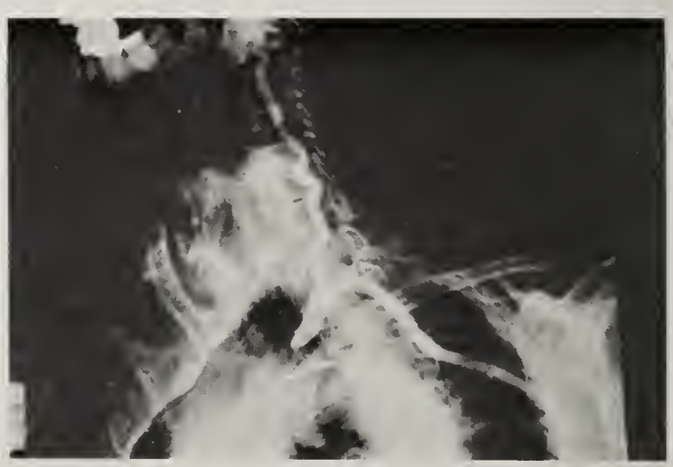


Figure 2

From the Department of Radiology, Scottsdale Memorial Hospital, 7400 E. Osborn Road, Scottsdale, Arizona 85251.

Surgery

Rupture Of The Gallbladder Due To Blunt Abdominal Trauma

D.C. Bullington, M.D.
W.F. Briney, M.D.

ABSTRACT

A case report of a ruptured gallbladder due to blunt abdominal trauma is given, with a review of the literature of this unusual event. It appears to be increasing in frequency worldwide, as the incidence of motor vehicle accidents rises, and merits consideration by all those caring for accident victims.

Blunt trauma to the abdomen is an ever increasing accompaniment of motor vehicle accidents. Isolated injuries of the gallbladder as a result of such blunt trauma are unusual but seem to be increasing. Norgore[1] reported on 32 cases in the literature from 1905 to 1946. Manlove et al[2] in 1959 indicated that only about 50 cases had been reported. Schechter[3] in 1969 in a comprehensive review of the subject cited about 110 authentic cases in the world literature and indicated an 8% occurrence among all gallbladder wounds proper. Rarenko[4] reported on 39 cases from his national literature in 1973 and gave an incidence of 1.6% in closed abdominal injuries. Therefore it appears that solitary blunt trauma to the gallbladder is an unusual event but due to high speed automobile accidents worldwide, the injury is occurring with greater frequency.

The injury itself is quite subtle and is often not suspected. Most cases are not diagnosed until the time of exploration. This is due to a combination of factors of which the following are recognized and discussed in the literature:[1-3,5,6]

(1) The protected location of the gallbladder under the liver and costal margin.
(2) Concomitant serious injuries drawing attention away from the area of the abdomen.
(3) Early lack of peritoneal reaction to sterile bile from ruptured normal gallbladders (the usual reported situation) in an otherwise obtunded victim due to alcoholic intoxication.

A high index of suspicion with prompt intervention is advisable, however, because of the high degree of mortality associated with the inevitable bile peritonitis. Most authors believe that the generalized soilage of the peritoneal cavity with concentrated gallbladder bile is highly lethal due to major shifts in body fluids and the subsequent onset of shock.[7,8] Infection plays an important secondary role and is a factor in late deaths. Localized bile peritonitis, on the other hand, may be tolerated for months.[1]

Etiological factors existing in traumatic rupture of the gallbladder include the relative ease of injury in a normal thin-walled gallbladder, especially one distended in the postprandial state or after excessive alcoholic intake.[7-9] A direct blow to the right upper quadrant of the abdomen is often the offending agent or there may be a history of a crushing injury or a decelerative impact from a steering wheel or a fall.[3,10]

There are no significant diagnostic features of an isolated traumatic gallbladder rupture. It may be suspected in an individual with a history of blunt trauma to the right hypochondrium with severe pain out of proportion to the evident injury, especially with pain referral to the right shoulder.[8] However, in less than 20% of cases is there an identifying mark on the skin.[10] Radiographic and laboratory studies offer little conclusive information. Multiple peritoneal taps or peritoneal lavage yielding bile-stained fluid are helpful but are not often obtained and are not diagnostic of

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gallbladder injury alone. There may be liver or biliary duct injury as well.[7] Intravenous cholangiography is mentioned by Fletcher[6] as being of possible value preoperatively but there are not sufficient reports to document its value.

The rate of the loss of bile into the peritoneal cavity is a factor, however. With smaller ruptures that wall off quickly, the clinical picture improves rapidly with appropriate resuscitative measures. When the bile enters the peritoneal cavity faster than it can be walled off, there is rapid progression of symptoms of an acute abdominal catastrophe.[3]

Some authors have classified gallbladder injuries into three groups: (1) contusion, (2) avulsion, and (3) laceration.[2,12] Penn[11] in 1962 added a fourth group, traumatic cholecystitis and Solheim[13] in 1972 described a fifth category, traumatic biliary peritonitis without rupture. Schechter[3] preferred to classify non-penetrating gallbladder injuries under two headings: (1) direct injuries such as bruising, perforation, laceration and avulsion, and (2) indirect injuries such as contrecoup, extension, hemocholecyst or hemocholedochus.

Therapy directed to this problem involves primarily cholecystectomy. Lesser procedures of cholecystostomy and simple closure and drainage are not recommended except in critically ill patients, especially in the face of incompletely defined damage to the biliary tract. A more definitive procedure should then follow when feasible. Operative cholangiography may aid in the diagnosis at surgery and, of course, a thorough exploration is mandatory. The ultimate goal should be the elimination of any source of actual or potential bile leakage and the preservation of adequate biliary drainage. This may have to be internal or external as demanded by the problem at hand.[3,10]

The following case exhibits several of the aspects mentioned above.

Case report: About 2 a.m. on 12 June 1977 a 49-year-old male was admitted to the hospital after being involved in an automobile accident. He had been the operator of the vehicle and sustained multiple injuries, including blunt trauma to the abdomen and an injury to the right knee. He was also acutely intoxicated, having consumed a large but unknown amount of alcohol. He complained of abdominal pain and pain in the right knee. Physical examination revealed a diffusely tender and silent abdomen; a superficial laceration of the forehead; and clinical evidence for a fracture of the right patella. Laboratory studies revealed a hemoglobin of 13.3 grams per 100 milliliters, a hematocrit of 39% and a white blood cell count of 14,600 cells per cubic millimeter with 67 segmented neutrophiles per cubic millimeter, 3 bands, and 26 lymphocytes per cubic millimeter. The urine contained 1+ proteinuria but was otherwise unremarkable. The serum amylase was 155 nephelos units (normal: 15 to 200 units). Radiographs revealed essentially normal findings with no free air in the abdomen and a comminuted fracture of the right patella. The acute intoxication obscured the clinical picture and a period of observation was felt to be warranted. A nasogastric tube was passed and an infusion of intravenous fluids was started. Several hours later he began to have more severe abdominal pain with referral to the right shoulder. The abdomen became boardlike, with rebound tenderness and a peritoneal tap revealed serosanguinous fluid with an amylase level of 72 nephelos units. The hematocrit level remained unchanged and vital signs were quite stable. Due to the increasing intensity of abdominal pain, exploratory surgery was felt to be indicated. At surgery, there was generalized bile contamination of the peritoneal cavity with a 0.2 cm. perforation of the fundus of the gallbladder which was draining bile freely. No other abnormalities were found in the abdomen, including the liver, biliary ducts, pancreas, spleen, stomach, small and large bowel. The patient had a cholecystectomy with drainage of the gallbladder bed and simple aspiration of the bile collections in the peritoneal cavity. Grossly and microscopically the gallbladder was normal except for the 0.2 cm. perforation in the fundus. A few days later the patient had a repair of the fractured patella and torn quadriceps tendon, and he then made a complete and uneventful recovery.

In summary, traumatic rupture of the gallbladder due to blunt trauma is an unusual event which is quite subtle in its early manifestations. Later, however, it leads to evidence of a severe intra-abdominal catastrophe but responds dramatically to prompt exploration and removal of the gallbladder. All those caring for acute automobile accident victims are urged to consider this problem in their initial assessment of the injuries sustained.

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Radiology — continued

From page 116

ANSWER — Subclavian Steal Syndrome

The major branches of the aortic arch are the innominate artery (which forms the right subclavian and right carotid arteries), the left carotid artery and the left subclavian artery. The innominate and left carotid arteries are well seen in Figure 1, but the left subclavian artery is occluded at its origin. A later film (Figure 2) shows that the left subclavian artery fills from retrograde flow in the left vertebral artery. The findings on the aortogram, in view of the patient's symptoms, are classic for the subclavian steal syndrome.

DISCUSSION

Occlusion of the proximal subclavian artery can cause reversal of flow in the ipsilateral vertebral artery, which "steals" blood from the cerebral circulation to supply the arm. The symptoms produced by this hemodynamic abnormality have been termed the subclavian steal syndrome.

Subclavian occlusion is two to three times more common on the left than on the right, and three times more common in men. Arteriosclerosis is the usual cause; thrombosis, tumor, Takayasu's arteritis, trauma and surgical procedure (e.g. Blalock shunt) may also produce subclavian occlusion. Neurologic symptoms consist of dizziness, vertigo and syncopal episodes. Peripheral symptoms are those of pain and numbness of the arm and hand, with increasing symptoms after exercise. Arm exercise may provoke neurologic symptoms, and most patients have a combination of neurologic and peripheral complaints. Physical findings include a significantly lower blood pressure in the affected arm (20 mm. Hg. or greater), and bruits over the neck. Angiography is the procedure of choice in evaluation of the subclavian steal syndrome, and the thoracic aortogram yields more information than selective studies.

Treatment of symptomatic subclavian steal syndrome is surgical. Extrathoracic approaches have advantages over intrathoracic exposure. Carotid-subclavian and subclavian-subclavian shunts have been used to re-establish antegrade flow in the vertebral, with the latter anastomosis being performed more often at present, since carotid artery flow need not be interrupted during the surgery.

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Special Article

Medicine In Mexico — VI Socialized Health System

Charles C. Daschbach
Sharon Don

The health service pattern in Mexico is an intricate spectrum with many continuities and contradictions. The socialized system of delivery is only one element which coexists with private medicine and traditional folk healers, all being affected by the early Aztec and Mayan cultures, the colonial Spanish Catholic Church, modern European social security ideas, North American public health concepts, and their own unique Latin American culture.

The *Secretaria de Salubridad y Asistencia* or SSA is the cabinet level ministry responsible for health and public welfare, overseeing an annual operating cost of 1.6 billion dollars or 16% of the national budget (1974 figures). The government's responsibility for the general welfare has its foundation in the 1917 Mexican Constitution with the premise that health and social well-being are a right, and not a privilege.

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TABLE 1
Mexican Health Services and Resources 1975-1976*

	Population Attended 1976	Pct.	Number† Physicians 1976	Pct.	Medical Facilities 1975 Without Beds	With Beds	Number of Beds
I. SOCIAL SECURITY INSTITUTIONS	20,511,657	32.9	22,896	43.2	1424	677	29,692
IMSS	15,815,646	25.4	11,660	22.0	466	408	19,781
ISSSTE	3,113,298	5.0	5,300	10.0	691	44	4,768
Armed Forces	650,000	1.0	1,484	2.8	71	187	2,815
PEMEX	520,000	0.8	1,113	2.1	119	13	694
National Railroad (FF.CC.)	412,713	0.7	694	1.3	77	25	1,634
II. GOVERNMENTAL SERVICES	31,064,594‡	50.0	11,766	22.0	380	1936	39,023
SSA			9,010	17.0	380	1816	25,042
Municipal and State			1,378	2.6	-	120	13,981
III. PRIVATE SECTOR†	10,658,290	17.1	18,338	34.6	104	1141	22,128
Free Enterprise Practice	9,224,719	14.8	11,660	22.0	-	1120	21,533
Others (private HMOs, Mexican Red Cross, etc.)	1,433,571	2.3	6,678	12.6	104	21	595
TOTAL	62,329,189	100.0	53,000	100.0	1901	3754	90,843

*From *Plan Nacional de Salud* 1974-1976 1977-1983 (3 vol.) Secretaria de Salubridad y Asistencia, Mexico, D.F., 1974 and *Educacion de Pregrado en Medicina y Enfermeria,* Subsecretaria de Planeacion (SSA), Mexico, D.F., 1977.
†Author's estimates from prorating various government documents (1973).
‡Approx. 20,000,000 persons of this figure lack permanent health care according to Mexican Government estimates.

In 1943 legislation created the *Instituto Mexicano del Seguro Social* or IMSS, an autonomous social insurance system, to deliver primary health care and social assistance to workers, financed by the insured workers, their employers, and the government. Originally designed to cover all workers in private industry, 15.8 million workers and dependents are currently registered with the IMSS, increasing by about 8% annually.

Both the SSA and the IMSS have evolved during the past 30 years into a model for other Latin American countries. Emphasis has been directed from curative services to preventive care, rural salary incentives have been instituted, and a unique system of medical audits has been developed. Also to insure quality care, there is a specified maximum case-load of patients per hour for each physician specialty. Cooperation between the IMSS, SSA, and the medical colleges permits over 50% of Mexican medical students and 80% of postgraduates to train in IMSS clinical facilities. All medical graduates must serve a six month to one year social service requirement under the supervision of the SSA in order to obtain a medical license. Recently, the SSA and the IMSS have focused their priorities on the rural sector, where an estimated 20 million Mexicans have no permanent health care.

As in other Latin American health care systems, different programs covering particular classes of workers have developed from the initial concepts. In 1960 Mexico created the Government Servants Social Security and Welfare Institute or ISSSTE in order to provide health care for all civil servants. Also included under special health programs (Table 1) are employees of *Petroleos Mexicana* (PEMEX), the National Railroad (FF. CC.), and the armed forces. A special joint coordinating committee attempts to integrate the activities of the SSA, ISSSTE, IMSS and these other services.

In comparison to the British national health program, Mexico's systems are of the same general type (social insurance, direct revenues) and have the same type of benefits (cash, comprehensive services). Large differences exist between the population covered (33% vs 100% in Great Britain) and in the source of funds. In Mexico the primary source is employer-employee taxes with the government paying 1.6% of earnings while in England, the principal source is government revenue (85%) aided by social insurance taxes and direct consumer cost (15%).

Mexico took a major step in national health planning in 1973 with her first national health convention and the formulation of a National Health Plan which will direct her socialized services through 1983. As a developing nation, her health concerns are those shared by both lesser developed and industrialized nations: rising costs, quality care, greater concern for prevention, and coverage of rural populations.

(Special thanks to Dr. Netzahualcoyotl Ruiz Gaitan, Jefe del Departamento de Planeacion y Estadisticas, SSA, for assistance with statistics in Table 1.)

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The Community In Family Medicine: A Synergistic Evolution

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INTRODUCTION

This paper will review the interactions of the disciplines of community medicine and family practice in a new medical school from years 1969 through 1976 at the University of Arizona Health Sciences Center. It will emphasize the synergistic interactions of community and family medicine at the undergraduate, graduate, postgraduate and allied health educational levels.

BACKGROUND

With the opening of Arizona's first medical school at the University of Arizona in Tucson, in 1969, the initial thrust of the school's fledgling Department of Community Medicine was to establish a neighborhood health center in a medically underserved area of the city. The emphasis for the center and the major objectives were community and service oriented.

Among the many issues faced were the nature and types of health personnel necessary in a primary care facility of this kind. This concern, coupled with the difficulty of recruiting from shrinking pools of primary care health manpower, spurred the interest of the department in assessing its role in developing primary care oriented personnel for the State of Arizona.

At the neighborhood health center the department focused on community outreach and delivery of primary care, while within the institutional walls of the medical school, the undergraduate training program focused on the teaching of clinical epidemiology and social determinants of health and illness to the medical students. A preventive medicine residency was established that concentrated its efforts in the area of health services administration and assisted local government to define the health needs of urban and rural Arizona.

Approximately two years after the initial thrust into the community, significant changes occurred that laid the foundation for the interactions of community medicine with family medicine. *First*, the department received approval to establish a family practice residency program. *Second*, as an outgrowth of the need to provide new types of health practitioners, training programs for physician's assistants and family nurse practitioners were developed. *Third*, based on the department's interest in community activities, the emergency service of the University Hospital was considered "the community hospital interface" and full responsibility was assigned to the department for its operation and management. *Fourth*, in the department's desire to be a further catalyst for the delivery of health care to rural communities, two medically underserved areas within fifty miles of Tucson were identified and active rural model health units were developed. *Fifth*, significant attitude changes were occurring at the undergraduate and postgraduate levels, focusing on the student interest in family practice as a clinically acceptable practice alternative to existing medical disciplines. *Sixth*, within the medical school the faculty voted to develop a new three-year variable timed curriculum with focus on interdepartmental teaching with the disciplines of family medicine and community medicine having significant undergraduate educational input.

With the above milestones in place, the name of the Department changed from Community Medicine to Family and Community Medicine and the interaction of the two became one in name. The remaining portion of this paper will discuss the interaction of community medicine with family medicine.

UNDERGRADUATE EDUCATION

Between the years 1969 and 1977, a significant change took place at the University in undergraduate medical education. The faculty voted in 1972 to initiate a three-year undergraduate medical curriculum. In essence this was a variable-time curriculum wherein a student could complete his/her undergraduate training in approximately 32 months or extend it to 48 months of training. During this period of academic change the Department of Family and Community Medicine became involved in numerous activities that focused on the relationship and intertwining of family medicine and community medicine. Prior to the student's matriculation in his first formal day of activities, the Department offered a series of Public Health Service (PHS) sponsored preceptorships. Students were assigned to group practices with family physicians; they became involved in state-wide, primary care programs and were assigned to independent study projects on culture, customs and the interactions of the psycho-social milieu with health care and health care delivery. As part of overall supervision of this initial student community involvement, family and community oriented physicians of the Department were the primary faculty. Students received their counseling, advice and direction from clinicians who wore for the first time in the medical school the label of family practitioner. With the entry into the first year of medical school and the new three-year curriculum, family physicians of the Department were not only involved in the undergraduate teaching programs of interviewing, physical diagnosis and patient assessment, but during the formative years the students were assigned to minipreceptorships to community based family physicians in primary care activities in Tucson. Teaching activities included the introduction of family studies which combined home visits by medical and nursing students to collect not only family-directed data, but community-oriented data. The blend of the data base of the family with the home visit, coupled with the assessment of the locale in which the family lived, provided a highly appropriate interaction of family medicine and community medicine. In a unit on community resources the focus was on what programs and activities the practicing family physician could call upon to meet the needs of his patients. This included a full scope of services both in underserved and overserved health care areas in Tucson and surrounding counties.

With the introduction of the problem oriented record by Dr. Weed.[1] and health hazard appraisal by Drs. Robbins and Hall,[2] the unique blend of prospective medicine and clinical epidemiology were focused by the faculty family physicians into the clinical setting. The "plan" with its components for more information, therapy, and patient education was now incomplete without assessment of the epidemiological risk factors that would assist the clinician in enabling the patient to reduce his overall health risk.

After the first fifteen months of the three-year medical school curriculum, the student entered his clinical clerkship

From: U of A Health Sciences Center, Tucson, AZ 85724.

rotations. The Department of Family and Community Medicine offered a family practice clerkship that was ambulatory oriented and had characteristics that reinforced the idea of interactions of community medicine with the primary care clinical practice of family physicians. The clinical clerkship offers continuous ambulatory care experience to the students. Included in the clerkship is a core content didactic experience that focuses on aspects of community medicine as well as clinical medicine.

While the emphasis was on the nurturing of the problem solving skills of the student in his first clinical year, the interactions of community with family practice was and is emphasized. For example, in the core content teaching day, a unit of instruction deals with community related topics including assessment of models of health care delivery, occupational medicine, and clinical preventive medicine. At the present time, for the last three days of the six-week clerkship, students are sent as a group to live and work in nearby communities in Arizona. We utilize the Morenci Mining Hospital, a self-contained community with health care facilities for a population of 8,000 people, operated by the Phelps-Dodge Corporation. Students visit the community, work in the hospital, make rounds with the attendings, have a series of seminars on occupational health problems as well as inspect the facilities with safety engineers.

During the last year of the undergraduate training program the students have a number of opportunities to combine community medicine with family medicine. Numerous special projects that include a clinical activity, predominantly family practice oriented, coupled with a research study component, are offered by the Department. These range from international health care programs, to ongoing assignments with the U. S. Public Health Service Indian Hospitals and field clinics in rural Arizona. The emphasis and blend is not entirely clinical and not entirely community but on those unique situations when the family physician practicing in underserved areas combines and uses the community resources and has knowledge of the community in order to function at an optimal level of patient care.

POSTGRADUATE EDUCATION

The Department of Family and Community Medicine offers residencies both in family practice and in general preventive medicine. At the present time, the bulk of resources and the majority of residents are in the family practice residency program. We currently accept eight physicians for each year of the family practice residency and two physicians for the residency in preventive medicine. While similar to other family practice residency programs in the United States, the University of Arizona offers a very distinct combined residency program in both family practice and community medicine. The combined program has attracted persons with interests both in clinical family practice as well as community medicine. Residents have extended their post-graduate training by a year, while spending elective time in community medicine, health care administration, epidemiology and maternal and child health. Family Practice Residents have been part of prospective epidemiological studies of Otitis Media among the Navajo as well as actively involved in training programs for Home Health Aids for use in our rural clinics. For those who select it, interaction at the residency level of Family Practice and Community Medicine, offers unique and expanded educational opportunities.

CONTINUING MEDICAL EDUCATION

In the development of our undergraduate and graduate training programs in family medicine and community medicine, it became obvious that while we were indeed focusing on the approximately fifteen percent of the young professional's total career in a formal academic setting, we were neglecting the needs of the family physician in practice. Continuing medical education has generally been thought to be the domain of an office of continuing medical education whose interests were primarily in meeting the requirements for formalized CME certification by medical societies, state legislatures and national organizations. With the requirement for recertification by the American Board of Family Practice, as well as AMA and ArMA Requirements in CME, the unique and special needs of the family physician in practice became of interest to the Department. It was the Department's feeling that, while residency and undergraduate training received a significant portion of the Department's resources, CME was a legitimate and mandatory area of service, research, and teaching interest.

Other factors influenced the movement into CME in the community. Among these were the need to identify community physicians who could be trained and function as preceptors for undergraduates as well as serve as the potential reservoir for associated clinical faculty for the residency program. In the CME arena, emphasis during the formative years of the program has focused on developing CME activities for the family physicians in the State of Arizona. Selected family physicians throughout the state serve as a sounding board for program format and content.

Numerous kinds of graduate training programs have been instituted. One type, the family practice review course occurs on an annual basis. As the Department of Family and Community Medicine is the prime agent responsible for development of these activities, the sensitivity of the unique needs of family physicians as well as the assessment of potential deficits in terms of use of community-related activities blend themselves well into the program format. While it is very appropriate to schedule a review of cardiovascular disease, it is also quite useful to look at the principles of health hazard appraisal and clinical epidemiology as it is related to cardiovascular disease. While topics concerning advances in the treatment of hypertension are timely, concurrently the question is discussed of the appropriate extent and value of the diagnostic workup for hypertension. The same questions are discussed in considering the "annual physical" and the family physician. The relationship of clinical epidemiology, health services and family practice easily blend.

In addition to review courses, monthly conferences on selected topics occur for family physicians around the state. Practicing physicians come to Tucson for a day and a half of intensive review. During these activities the informal social gatherings is important. Needs and requirements of program developments and methods of health care delivery in the settings in which our residents will practice and the family physicians operate are shared attentively between the faculty and the course participants.

A community scholar program has been initiated for family physicians to return to the department for a week, join in rounds, academic teaching exercises, and informal discussions in a framework of activities designed to meet his/her perceived and verbalized needs. This offers the opportunity for the Department to maintain an active interest in the community of family physicians, and their individual needs and enables the faculty to bring directly to the residency the "real world" dimension of the physician's community life and professional responsibility for his CME.

ALLIED HEALTH

With its interest in primary care, it was a natural extension of the Department concurrently to become involved in training of physician extended manpower for the state. As the Department developed experience with family practice, ambulatory care, outreach services, rural health, neighborhood health movement, the supply of other forms of health manpower to meet perceived and expected needs was required.

In Arizona, with its significant Indian population distributed over wide areas, requirements for primary care, health assessment, emergency evacuation, preventive services, out-patient care and the need to develop and expand outreach

services to help solve these community problems was necessary. In conjunction with the U. S. Public Health Service, the Department has assisted in the training and evaluating of Indian Community Health Medics. The CHMs are now assigned throughout the U. S. and in Alaska as extension of family physicians in the U. S. Public Health Service health care facilities. This activity in the main has been supported by the Family Practice component of the Department.

In addition to training health personnel for the U.S. Public Health Service program, the Department of Family and Community Medicine has been actively involved in training family nurse practitioners for use in the State of Arizona. Family Nurse Practitioners are now used in public health clinics, categorical health programs, such as tuberculosis, family planning and maternal and child health, as well as general physician extenders for urban and rural and underserved areas of Arizona. This has been one of the attempts to interact and bridge the community medicine component with the family medicine structure at the University. Presently, two nurse practitioners are part of the faculty at the Family Practice Model Unit.

SUMMARY

In summary, this paper has demonstrated the spectrum of activities where Community Medicine and Family Medicine interact in the university setting. The context in which the family physician provides service and care is the community. Knowledge of the community, whether a group of families, a geographical area, or an on-going specific cohort of individuals, is also the responsibility of the newly-defined family physician. It is our observation that the interaction of community with family medicine, and the sensitivity of the family physician to the needs, the resources, and the constraints of the community are all closely intertwined. The development of the educational program in the Department of Family and Community Medicine at the University of Arizona over the past six years suggests that the interactions of community medicine with family medicine is a significant and viable relationship in which both disciplines have the capability of growth in a synergestic relationship.

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U of A Grand Rounds

Pathophysiology And Therapy Of Reflux Esophagitis

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Introduction

The concept of a functional lower esophageal sphincter was first formulated to explain the absence of reflux of gastric contents into the esophagus in normal people. In the normal resting state intragastric pressure is positive by 10-30 mm Hg and intrathoracic pressure is negative by 3-9 mm Hg. This would normally create a gradient which would favor nearly constant reflux of gastric contents into the esophagus. There have been numerous hypotheses to explain the lack of reflux in normal people and its presence in people with symptomatic reflux esophagitis. In this paper the current concepts of the pathophysiology of reflux esophagitis are reviewed. These concepts form the basis for a rational approach to therapy.

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Table I

Autonomic Drugs Which Alter LESP

Increase LESP	Decrease LESP
1. Cholinergic (muscarinic) bethanechol*, methacholine*	1. Ganglionic stimulants nicotine*, DMPP
2. Anticholinestrerase edrophonium*	2. Atropine
3. Alpha-adrenergic norepi*, phenylephrine*	3. Beta-adrenergic epinephrine*, isoproteronol*
4. Beta adrenergic blockers	4. Alpha-adrenergic blockers
	5. Dopamine

**Indicates those which have been studied in man.*

Table II

Hormones Which Alter LESP

Increase LESP	Decrease LESP
1. Gastrin*, Pentagastrin*	1. Secretin*
2. Caerulein*	2. Cholecystokinin*
3. Bombesin	3. Glucagon*
4. Motilin	4. Progesterone*
	5. Vasoactive Intestinal Peptide*

**Indicates those which have been studied in man.*

Current Concepts of Esophageal Reflux

Although there is no evidence to support the concept of an anatomic lower esophageal sphincter, there is now much evidence documenting the existence of a physiologic spincter, i.e. a high pressure zone, 1-4 cm long in the distal esophagus.[1] Pressures are higher in this zone than in the adjacent esophagus or stomach. This high pressure zone which is 12-35 mm Hg higher than intragastric pressure maintains a closed lower esophageal segment except during swallowing when pressure decreases to the level of intragastric pressure and the segment opens to allow passage of a liquid or solid bolus propelled by gravity and esophageal peristalsis.

The most consistent physiologic finding in patients with reflux esophagitis is the presence of a reduction in lower esophageal sphincter pressures (LESP) compared to normals.[2] Measurement of gastric and LESP in normal people using a perfused catheter connected to a pressure transducer shows an average resting sphincter pressure of 15.0 ± 1.7 mm Hg above gastric pressure. Patients with gastroesophageal reflux have resting sphincter pressures of only 7.3 ± 0.5 mm Hg above gastric pressure.[3] Lower esophageal sphincter pressure is probably not the only determinant of the presence or absence of gastroesophageal reflux since most patients with reflux esophagitis still have a sphincter pressure which is higher than intragastric pressure even during physical activities which increase intragastric pressure.[4] In addition, although some studies show a clear separation of sphincter pressures in normal people and those with esophageal reflux, other studies show significant overlap of sphincter pressures in the two groups.[2] In spite of these considerations most recent studies conclude that the LESP is the most important barrier to gastroesophageal reflux.

Determinants of LESP

Mechanical Factors

A mechanical factor which appears to influence LESP is the length of the intraabdominal segment of esophagus.[5] Although hiatus hernia alone is not sufficient to result in reflux esophagitis,[6] many hiatus hernia patients who have surgical restoration of an intraabdominal segment of esophagus (Hill repair) will have an increase of LESP.[7] However, in human and animal studies an increase in LESP correlates only weakly with the length of the intraabdominal segment of esophagus.[7] Many patients with esophageal reflux

Table III

Miscellaneous Substances Which Increase LESP

Drugs	Local "Hormones"	Food
1. Betazole*	1. Histamine (H1 receptor)	1. Protein*
2. Metoclopramide*	2. Serotonin	
	3. Prostaglandin $F_{2}a$	

**Indicates those which have been studied in man.*

have no anatomic displacement of their LES; and conversely many patients with hiatus hernia have normal LESP and no reflux.[6]

Neural Factors

The major neural control of esophageal sphincter tone is via the vagus nerve.[8] The primary result of vagal stimulation is relaxation of the LES via non-cholinergic, non-adrenergic fibers. The vagus also contains afferent fibers which respond to distension of the esophagus and result in reflex increase in LESP. In addition, these afferent fibers may mediate the increase in LESP which is seen in response to increased intragastric pressure with straining or Valsalva Maneuver. Neural control of the LES may involve many other pathways in addition to the vagus nerves. It is also well documented that various autonomic drugs produce either an increase or decrease in LESP (See Table I).

Hormonal Factors

The precise role of hormones in controlling LESP is still the subject of much study. Numerous hormones have been shown to either decrease or increase LESP in man and in animals when administered in supraphysiologic doses[1,9] (See TableII). The role of most hormones in modulating LESP is controversial. However, there is recent evidence to suggest that the frequent occurrence of "heartburn" in pregnancy may be related to a progesterone effect.[10] A recent study in normal pregnant women showed a progressive decrease in LESP as term was approached. The decrease correlated well with increasing serum progesterone concentrations. Reduction of LESP was also noted in a group of normal women during ingestion of an estrogen-progesterone oral contraceptive but not during estrogen administration alone.[11] These studies suggest that progesterone may cause or exacerbate reflux esophagitis.

Table IV

Miscellaneous Substances Which Decrease LESP

Drugs	Local "Hormones"	Food
1. Dipyridamole	1. Histamine (H2 receptor)	1. Chocolate*
2. Nitrites*	2. Prostaglandins (E_1, E_2*, A_2)	2. Caffeine*
3. Theophylline		2. Peppermint*
4. Ethanol*		
5. Nicotine (smoking)*		

**Indicates those which have been studied in man.*

Miscellaneous Biochemical Factors

There are numerous drugs, endogenously produced tissue substances and foods which have been reported to produce either an increase in LESP (See Table III) or a decrease in LESP[1,12,13] (See Table IV). The precise role of the endogenously produced tissue substances, e.g. prostaglandins, and of various foods in modulating LESP under physiologic conditions remains to be determined. However the known effects of some of the substances, especially the food items and drugs, suggests various therapeutic maneuvers which may be of value in treating reflux esophagitis.

Intrinsic Myogenic Factors

As discussed above, there are numerous factors which appear to modulate LESP. There is recent evidence to suggest that LESP is primarily a function of intrinsic myogenic factors, i.e. intrinsic muscle tone.[14] These studies suggest that the basic defect in

people with reflux esophagitis and reduced LESP may be related to an intrinsic defect in the muscle of the LES. This possibility does not negate the importance of the previously mentioned factors which may also influence LESP and consequently the tendency to esophageal reflux.

Therapy of Reflux Esophagitis

Medical Therapy

The medical therapy of reflux esophagitis is outlined in Table V. Certain aspects of the therapy require emphasis. A potent non-calcium containing antacid should be used in adequate doses.[15] For patients with moderately severe symptoms of reflux esophagitis 30 cc of a magnesium hydroxide-aluminum hydroxide combination (e.g. Maalox or Mylanta II) should be taken one and three hours postprandially and 60 cc at bedtime. Eating three regular meals per day is an important part of this regimen. The use of calcium containing antacids on a regular basis results in at least mild renal functional impairment and an acid rebound phenomenon which results in significant increase in gastric acid output. The use of an alginate containing antacid (e.g. Gaviscon) has not been shown to be more efficacious than the antacid regimen outlined above The H_2 antagonist cimetidine produces up to a 90 percent reduction in basal gastric acid output and a 60-80 percent reduction in meal stimulated acid output.[16] There is no current evidence that cimetidine is either more or less effective than the above antacid regimen. However it may be preferable for some patients because of diarrhea with high dose magnesium hydroxide-aluminum hydroxide antacids. In addition, cimetidine is an appropriate alternative for chronic renal failure patients in whom potent magnesium containing antacids are contraindicated. Cimetidine offers the additional advantages of long duration of action (4-6 hours) compared to antacids (1-2 hours) and may be preferable for h.s. treatment.

The mechanical means of reducing gastroesophageal reflux which are outlined in Table V are self-explanatory. It should be emphasized that elevation of the head of the bed should be done with blocks under the head of the bed and not with wedges under the mattress or simply extra pillows. The latter two methods of elevation tend to increase intraabdominal pressure and therefore gastroesophageal reflux.

Table V

Medical Therapy of Reflux Esophagitis

A. Neutralize acid
 1. Antacids
 2. Antacid-Alginate (Gaviscon)
B. Decrease acid output
 Cimetidine
C. Decrease reflux mechanically
 1. Elevate head of bed 30°
 2. Avoid reclining for 3-4 hours after eating
 3. Avoid large meals
 Avoid heavy activity after meals
D. Restore LESP toward normal
 1. Dietary measures (avoid fats, chocolate, peppermint)
 2. Avoid smoking
 3. Avoid drugs which decrease LESP
 4. Gastric alkalinization—antacids or cimetidine
 5. Bethanecol (Urecholine)
 6. Metoclopramide (not yet released for use)

The average LESP over a period of 24 hours may be restored to higher, although usually not normal levels, with bethanechol (Urecholine) 25 mg q 6 h.[17] The frequency of symptomatic esophageal reflux has been significantly reduced in controlled trials with this drug. Metaclopramide which is a drug chemically related to procainamide has been shown to directly stimulate smooth muscle contraction. It produces an increase in LESP and a significant decrease in symptoms of reflux esophagitis.[17] It has none of the toxic effects of procainamide. It is not yet approved for use in this country, but is available in Mexico.

Table VI

Indications for Surgical Therapy Of Reflux Esophagitis

A. Intractable symptoms on intensive medical therapy
B. Recurrent bleeding esophagitis
C. Esophageal stricture of short duration
D. Repreated evidence of aspiration

Surgical Therapy

There are no absolute criteria for recommending surgical therapy for reflux esophagitis. The current generally accepted indications for an antireflux procedure are outlined in Table VI. Intractable symptoms should be associated with endoscopic verification of moderate to severe esophagitis before surgery is done. Endoscopic verification of bleeding from esophagitis should also be obtained prior to considering surgery for that condition. Strictures which have been present for longer than 2-3 years are often difficult to treat surgically and surgery should probably not be done for this reason unless severe pain, bleeding or aspiration episodes are present or esophageal bougie dilitation of the stricture is ineffective.

Surgical treatment of esophageal reflux involves creation of a long intraabdominal segment of esophagus with either an invagination of the esophagus into the stomach or a fundoplication in which the fundus of the stomach is wrapped around the intraabdominal segment of esophagus. These procedures result in an average increase of 10-12 mm Hg in LESP and produce significant or total relief of symptoms in 85-96 percent of patients[7] when performed by an experienced surgeon.

Summary

Although the primary defect which results in reflux esophagitis has not been unequivocally established, the bulk of the evidence favors a primary esophageal muscle defect. An understanding of the numerous factors which may modify LESP assist in planning vigorous medical management. When medical management fails to prevent severe symptoms or complications then surgery should be considered. Current surgical techniques have resulted in very satisfactory control of symptoms in most patients with inadequate response to medical therapy.

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Con

The Arizona Hospital Association's ad hoc Committee on HR 6575 alternatives and the Arizona Medical Association's Health Care Cost Committees have separately and jointly diligently studied the multiple ramifications involved in attempting to contain and control the expenditure of Health Care dollars. Both committees have prepared reports which agree on many, many philosophical issues; on many, many of the problems involved; and, on many, many possible solutions to some of the problems. In fact, it can truthfully be stated that the positions of the two committees and their parent organizations are completely compatible except in the area of Certificate of Need (CON).

Because of legislation, the hospitals of Arizona have for some time been under CON regulation. The Legislature, in their wisdom, felt regulation was necessary to prevent over-building and unnecessary competition in certain areas of Health Care Services. Hopefully CON regulations would produce a more orderly growth in the affected areas. Expenditures by physicians was specifically left untouched by CON by the Legislature.

Consequently, some of the hospitals have quite justifiably felt this represented a basic unfairness in the law when expensive diagnostic equipment such as CT-scanners began to show up in physicians' offices. The added burden of expense and hassle involved in obtaining the CON is borne only by the hospitals and puts them at an immediate financial disadvantage. Exemption from the CON process by the physician might also at times interfere with orderly and supervised expansion of Health Care Services.

The present Certificate of Need law definitely needs some revisions and reworking to correct problems it has created. In the process of accomplishing this, some of the hospital people have felt that the CON regulations should also apply to physicians' offices for the above stated reasons. The position of ARMA has always been to oppose CON as it pertains to physicians' offices.

In spite of the "what's fair is fair" argument to which we all subscribe in principle, ARMA's position of opposition should remain unchanged for a number of reasons. First of all, the CON application process can involve thousands of dollars which is a much greater burden comparatively to a physician than a hospital. Secondly, the time involved in the various bureaucratic hearings and similar gyrations occur during the bureaucratic working day. The hospital has plenty of administrative personnel to attend during their working day, while the physician has to take time away from patient care to attend — which in itself is a self-defeating exercise.

The current issue has arisen largely because of the CT-scanner. The production and sales of this type of equipment is highly competitive and the entire field changing rapidly. Used full-body scanning units are available in the $200,000 category. New headscanning units are available for less than $100,000 which is considerably less than ordinary diagnostic x-ray equipment. Because of this, more and more units will find their way into usage. Hopefully this reduction in price may be reflected by lowering of the cost of service to the patient.

Considering the patient, the cost of the CON application is obviously passed on to the patient. Additionally, the current CON laws have been shown by David S. Salkever, Ph.D. of the Johns Hopkins University and Thomas W. Bice, Ph.D. of Washington University in St. Louis to "encourage certain types of investments that in the long run increase the cost of hospital care by a reduction of capital expenditures from new beds to new services and equipment." Clearly, CON adds to patient care costs which we are all trying to contain.

Obviously both organizations, on behalf of reduced Health Care costs, need to take the CON issue back to the Legislature for re-examination. Something needs to be worked out to voluntarily limit investment in high-cost equipment in geographical settings that are certain to produce unnecessary duplication of service. We, as physicians, need to educate ourselves, our patients, and our friends in the Legislature on this important issue in hopes of obtaining a reasonable and workable consensus which will decrease, rather than increase, costs to our patients.

J.F. Kahle, M.D.,
President, ArMA

The Arizona Specialized Center Of Research In Pulmonary Disease

In 1971, the Specialized Center of Research (SCOR) in Pulmonary Disease was established at the University of Arizona under a grant from the National Heart, Lung, and Blood Institute. This is a multidisciplinary approach to the problem of obstructive lung disease involving members of several departments in the College of Medicine. A major project undertaken by the SCOR has been a prospective longitudinal study of respiratory health in a random sample of Tucson households with the specific objectives of elucidating the natural history of, and determining the factors contributing to, the development of chronic obstructive lung disease.

Over the past five years, yearly data collected on this population of 4000 people have included extensive health information from self-administered questionnaires, spirometric flow-volume data, results of closing volume tests, vectorcardiograms, atopic skin test reactivity, analysis of blood samples for alpha-1-antitrypsin deficiency, and, in the current survey year, chest roentgenograms and helium-oxygen vs. air flow-volume curves. Based on these data, normal values have been determined for various spirometric, flow-volume, and closing volume parameters and these parameters tested for sensitivity in detecting abnormalities in population subgroups defined as other than normal. The closing volume test, originally thought to be a sensitive index of early obstructive disease, proved not to be so when applied to a general population. People who are heterozygotic for alpha-1-antitrypsin deficiency (based on trypsin inhibitory capacity measurements and on phenotype determination) were found to have no greater prevalence of respiratory symptoms, disease, or of abnormal lung function than the remainder of the population, though additional studies of this genetic factor are currently in progress. Subjects who had a history of respiratory trouble before 16 years of age, however, do have a higher prevalence of adult obstructive disease and an accelerated decline in lung function than those without such a history. Whether this is a cause-effect phenomenon or simply a manifestation of increased susceptibility to respiratory trouble present throughout life is under investigation. In adults, frequent disabling acute respiratory illness often precedes the onset of newly diagnosed obstructive disease. Among subjects without a history of childhood respiratory trouble, a dose-response effect of cigarette smoking can be demonstrated, revealed by accelerated decline in lung function with age. The effects of aging alone on the mechanical properties of the adult human lung have been clarified in a special study of men and women between the ages of 25 and 75 years in whom rigorous selection criteria were applied to exclude the effects of disease, injury, or genetic factors. Knowing how the normal lung is affected by age alone will allow us to evaluate the impact of these other factors.

Though obstructive disease, in its various forms, is extremely common, little has been known about how it develops or why some, but not all, people appear likely to be afflicted. In the five years of the Arizona SCOR study, much has been learned. It is anticipated that these 4000 people will be followed for another five to ten years and that, as a result of this longitudinal study, knowledge will be gained which can be applied to reducing the impact of this major public health problem.

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Auxiliary Highlights

OF, BY, and FOR are the three little words relating to Auxiliary what and wherefore. Trite, perhaps, but true.

Auxiliaries OF the counties produce great efforts BY individual members FOR the medical profession's goal of health improvement.

To achieve purpose as reflected by aims of the profession, the medical Auxiliary has developed an umbilical lifeline from National headquarters in Chicago to organized county auxiliaries and members at large.

Regional workshops provide localized enthusiasm each year for state chairmen who attend by virtue of currently selected priorities. The chairmen in turn relay the message via state workshop, sometimes traveling to the counties to do so.

The Chicago Confluence offers excellent orientation for state presidents-elect, and state presidents share achievement reports at conventions. Communications move all directions among National, state, and county in the important emphasis of getting acquainted in small groups and working together with National.

The Arizona Auxiliary, ArMA's counterpart helpmate, works in doctor-approved direction and offers guidance for the broad scope of effort which is spent in the counties. Results come as this most effective use of people power is utilized where the needs and problems exist.

Arizona Auxiliary boasts a heritage of full-line participation even including a National president. Indeed, Arizona stars appear regularly in the nationwide tapestry of Auxiliary supporters of medicine. "Stars" and "garters" (!) one might say; has your spouse joined yet?

Mrs. M.W. (Jean) Phillips
Auxiliary Editor

Not Listed In The AMA

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

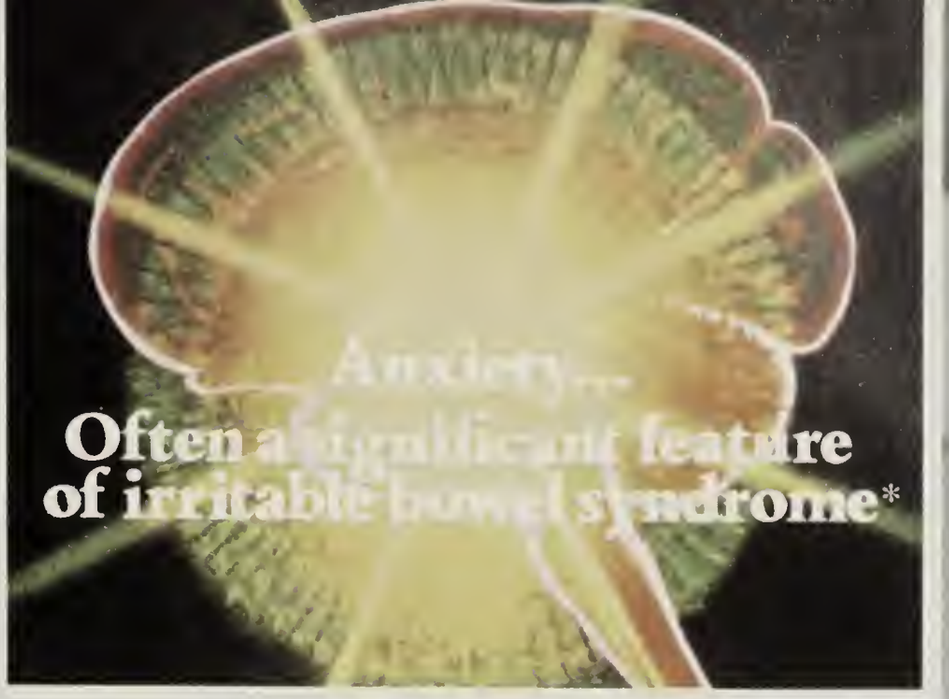
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Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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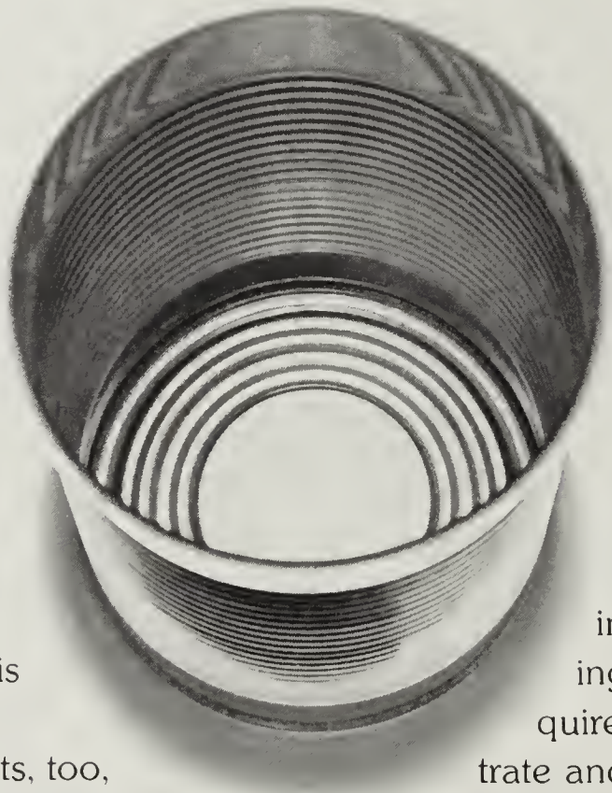
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ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

MEDICAL EDUCATION COMMITTEE

Meeting of the Medical Education Committee of the Arizona Medical Association, held Thursday, September 22, 1977, at 810 West Bethany Home Road, Phoenix, convened at 7:05 p.m., Robert E.T. Stark, M.D., Chairman, presiding.

SECTION REPORTS

Section on Accreditation

Dr. Sheeley reported that the Section on Accreditation recommended approval of the accreditation of the CME program of the Arizona Heart Institute.

IT WAS MOVED AND CARRIED TO ACCEPT THE RECOMMENDATION OF THE SECTION ON ACCREDITATION THAT THE ARIZONA HEART INSTITUTE'S CONTINUING MEDICAL EDUCATION PROGRAM BE APPROVED FOR A PERIOD OF TWO YEARS, WITH RE-SURVEY AT THE END OF THAT PERIOD.

Dr. Hastings submitted proposed revised accreditation materials as extracted from several sources, to be assembled by staff and submitted to the Section for approval.

Section on Certification

Dr. Taylor submitted requests from two physicians who have asked for special consideration on meeting their CME requirements, due to illness and consequent inability to attend meetings. Dr. Taylor recommends that these physicians not only submit a list of their medical reading, but also a written review of their reading. Each year they will be asked to submit a review on a topic of their choice as evidence of the continuing medical education merits of their reading. This review should include a bibliography, references, discussion of pros and cons, etc.

IT WAS MOVED AND CARRIED THAT THE SECTION ON CERTIFICATION BE DELEGATED TO APPROVE CME ACTIVITIES OF PHYSICIANS WHOSE ILLNESS MAKES IT IMPOSSIBLE FOR THEM TO MEET THE REQUIREMENTS IN THE USUAL MANNER, AND THAT THE CME STANDARD FOR SUCH PHYSICIANS SHALL BE A DEGREE OF ACTIVITY WHICH EQUATES TO THE FIFTY-HOUR PER YEAR REPORT.

Section on Graduate Medical Education

Dr. Layton reported on recent meetings of the American Board of Medical Specialties, and developments which are taking place in Graduate Medical Education, the role of the Liaison Committee on Graduate Medical Education, the Coordinating Council on Medical Education, the Residency Review Committees, etc., as well as formation of a new committee established by the Secretary of Health, Education and Welfare which is looking at present and future supply of and requirements for physicians, examining specialties and geographical distribution, etc. This Committee, the Graduate Medical Education National Advisory Committee (GEMENAC) has met once and is expected to become fairly active.

Section on Requirements

Dr. Wagner submitted recommendation of the section that the request of the A.R.E. Clinic for CME credit in Category 1-d for their January, 1978 symposium be denied.

IT WAS MOVED AND CARRIED TO SUPPORT THE RECOMMENDATION OF THE SECTION ON REQUIREMENTS REGARDING THE REQUEST FROM THE A.R.E. FOR CME CREDIT.

CONFERENCE OF STATE CME CHAIRMEN

Dr. Stark reported on correspondence and proposed by-laws, etc., concerning the subject organization. The Committee voted to support continued participation by ArMA in this group.

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

THE FOLLOWING INSTITUTIONS AND ORGANIZATIONS HAVE RECEIVED ACCREDITATION FOR CONTINUING MEDICAL EDUCATION FROM THE COUNCIL ON MEDICAL EDUCATION OF THE AMA

ARIZONA MEDICAL ASSOC., INC.
ARIZONA STATE HOSPITAL, PHOENIX
DESERT SAMARITAN HOSPITAL, MESA
GOOD SAMARITAN HOSPITAL, PHOENIX
HEALTH MAINTENANCE ASSOCIATES
PHOENIX INDIAN MEDICAL CENTER
PHOENIX PSYCHIATRIC COUNCIL
MARICOPA COUNTY GENERAL HOSPITAL, PHOENIX
MEMORIAL HOSPITAL, PHOENIX
ST. LUKE'S HOSPITAL AND MEDICAL CENTER, PHOENIX
ST. JOSEPH'S HOSPITAL AND MEDICAL CENTER, PHOENIX
TUCSON HOSPITALS MEDICAL EDUCATION PROGRAM, TUCSON
U OF A HEALTH SCIENCES CENTER
VETERANS ADMINISTRATION CENTER, PRESCOTT
VETERANS ADMINISTRATION HOSPITAL, PHOENIX

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category I credit. Physicians who participate in programs which are designated Category I by accredited institutions will receive Category I credit toward the ArMA Certificate in CME and the AMA's Physicians Recognition Award.

MARCH

COMBINED GROUND ROUNDS

March 1, 1978, St. Joseph's Hospital. Sponsor: Family Practice Residency Programs of St. Joseph's, Good Samaritan, and Scottsdale Memorial Hospitals. Contact: Ann Larsen, R.N., Program Coordinator, Family Practice Residency Program, St. Joseph's Hospital and Medical Center, Phoenix, AZ 85013. Approved for required hours toward the AMA Certificate in Continuing Medical Education.

VALLEY OF THE SUN DERMATOLOGIC SYMPOSIUM

March 2-5, 1978. Casa Blanca Inn, Scottsdale, AZ. Sponsor: Phoenix Dermatological Society, & University of Arizona. Contact: Dr. Fillmore K. Bagatela, Dr. Peter J. Lynch, 333 E. Virginia, Phoenix, AZ. Approved for 12 required hours toward the ArMA Certificate in Continuing Medical Education.

DAY OF HEMATOLOGY

March 3, 1978 Camelback Inn, Scottsdale AZ Sponsor St Joseph's Hospital and Medical Center Phoenix AZ Contact Ethelann Murray, M D , St Joseph's Hospital and Medical Center 350 West Thomas Road, Phoenix, AZ 85013 Approved for 6 required hours toward the ArMA Certificate in Continuing Medical Education

THE SECOND NATIONAL CONFERENCE ON OTITIS MEDIA

March 3-5 1978. Doubletree Inn, Scottsdale AZ Sponsor Univ of Arizona College of Medicine and the Indian Health Service Contact Office of Continuing Medica Education and Outreach University of Arizona College of Medicine, Tucson, AZ 85724 Approved for 17 5 required hours toward the ArMA Certificate in Continuing Medical Education

NEW MEXICO METABOLISM SYMPOSIUM

March 9, 10 & 11, 1978. Taos, New Mexico. Sponsor: Taos, New Mexico. Sponsor: Taos Metabolism Symposium. Contact: Office of Education, Lovelace-Bataan Medical Center, Albuquerque, New Mexico.

PURGATORY COLORADO MEDICAL SKI SYMPOSIUM. "GERIATRICS: Diagnosis and Treatment".

March 10-11, 1978. Durango, Co. Sponsor: New Mexico Academy of Family Physicians. Contact: Albuquerque Academy of Family Physicians, 2650 Yale, S.E., Albuquerque, N.M. 87106. Approved for 4 required hours toward the ArMA Certificate in Continuing Medical Education.

CURRENT CONCEPTS IN KIDNEY DISEASE

March 10, 11, 1978, Safari Resort & Convention Center, Scottsdale, AZ Sponsor: Department of Medicine, Maricopa County General Hospital, Phoenix. Contact: Myra DaBolt, Adm. Assist., Maricopa County General Hospital, Phoenix, AZ 85008. Approved for nine required hours toward the ArMA Certificate in CME & AMerican Academy of Family Practice.

2ND ANNUAL SEMINAR, CURRENT CONCEPTS OF HYPERTENSION.

March 11-12, 1978. Scottsdale Registry Resort, Scottsdale, AZ. Sponsor: Health Maintenance Associates. Contact: Dr. L. A. Oslen, Health Maintenance Associates, 4747 N. 22nd St., Phoenix, AZ 85016. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

CURRENT CONCEPTS IN CEREBRO-VASCULAR DISEASE.
March 11-18, 1978. Sun Valley Inn, Sun Valley, Idaho. Contact: Margo Walter, Administrative Assistant, Office of Continuing Medical Education and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 22.5 required hours toward the ArMA Certificate in Continuing Medical Education.

GASTROENTEROLOGY FOR CLINICIANS — 1978
March 13-15, 1978. Del Webb's Towne-House, Phoenix, AZ. Sponsor: American College of Gastroenterology & the Institute of Gastroenterology, Good Samaritan Hospital. Contact: David C. H. Sun, M.D., Good Samaritan Hospital, Phoenix, AZ 85036. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

"MANDATORY REPORTING OF CHILD ABUSE"
March 18, 1978, Arizona Medical Association offices, Phoenix. Sponsor: Psychiatric Services, Ltd. Contact: Robert T. Dean, Jr., M.D., Ste. 121, 2202 E. Camelback Rd., Phoenix, AZ 85016.

CLINICAL RECOGNITION AND MANAGEMENT OF HEART DISEASE 1978
March 23-25, 1978. Auditorium, AHSC, Tucson, AZ. Sponsor: Dept of Cardiology, and Internal Medicine, U of A Health Sciences Center. Contact: Frank I. Marcus, M.D., 1501 N. Campbell, Tucson, AZ. Approved for 20 required hours toward the ArMA Certificate in Continuing Medical Education.

14th ANNUAL ARIZONA CHEST SYMPOSIUM
March 31, April 1, 2, 1978. Doubletree Inn, Tucson, AZ. Sponsor: U of A Health Sciences Center, Tucson Medical Center. Contact: Charles E. Blair, M.D., Carl F. Diener, M.D., Tucson Veterans Administration Hospital, Tucson, AZ. Approved for 20 required hours toward the ArMA Certificate in Continuing Medical Education.

APRIL

ISSUES IN CRITICAL CARE MEDICINE
April 8, 1977. Arizona State University. Sponsor: Desert Samaritan Hospital, Contact: L. A. Rosati, M.D., Dept. of Path., Desert Samaritan Hospital. Approved for 7 required hours toward the ArMA Certificate in Continuing Medical Education.

GERIATRIC POW-WOW
April 7, 8, 9, 1978. Arizona Inn, Tucson, AZ. Sponsor: Continuing Medical Education, U of A. Contact: A. Vuturo, M.D. Office of Continuing Medical Education and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 16 required hours toward the ArMA Certificate in Continuing MedicEl Education.

PEDIATRIC RADIOLOGY FOR THE GENERAL RADIOLOGIST
April 14-16, 1978, Doubletree Inn at Scottsdale Mall, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contact: A.R. Sandrock, M.D., Chairman, Dept. of Radiology, Maricopa County General Hospital, 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 15 required hours toward the ArMA Certificate in Continuing Medical Education.

ARIZONA MEDICAL ASSOCIATION 86th ANNUAL CONVENTION.
April 25-29, 1978, Tucson Community Center and Marriott Hotel, Tucson, AZ. Sponsor: Arizona Medical Association, Inc. Contact: Mr. Bruce E. Robinson, 810 W. Bethany Home Rd. Phoenix, AZ 85013. Approved for required hours toward the ArMA Certificate in Continuing Medical Education. Detailed program will appear in the March issue of *Arizona Medicine.*

MAY

UPDATE: PRIMARY CARE 1978
May 3-6, 1978. Arizona Health Sciences Center Sponsor: U of A Continuing Medical Education. Contact: A. Vuturo, M.D., Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 25 required hours toward the ArMA Certificate in Continuing Medical Education.

RURAL HEALTH/EMERGENCY CARE CONFERENCE
May 12, 13, 1978. Little America, Flagstaff, AZ. Sponsor: U of A Continuing Medical Education Program. Contact: John Hughes, M.D., Emerg. Serv. Dept., or Anthony Vuturo, M.D., Continuing Medical Education and Outreach, U of A Health Sciences Center. Tucson, AZ 85724. Approved for 12 required hours toward the ArMA Certificate in Continuing Medical Education.

JUNE

17th ANNUAL STATE CONVENTION
Maricopa Chapter, American Association of Medical Assistants, June 9, 10, 11, 1978, Doubletree Inn, Scottsdale, AZ. Sponsor: Maricopa Chapter, American Association of Medical Assistants. Contact: Jan Neal, CMA Convention Chairman, 4502 N. 36th St., $108, Phoenix, AZ 85018.

MEDICAL HORIZONS: ADVANCES IN THERAPEUTICS
June 30-July 2, 1978. Little America Hotel, Flagstaff, AZ. Sponsor: Office of Continuing Medical Education, U of A Health Sciences Center, Tucson, AZ. Contact: Anthony F. Vuturo, M.D., AHSC, Tucson, AZ 85724. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

JULY

21st ANNUAL RUIDOSO FAMILY PRACTICE SEMINAR
July 17-20, 1978. Headquarters Inn, of the Mountain Gods at Mescalero, New Mexico. Sponsors: New Mexico Chapter of the American Academy of Family Physicians. Contact: Bob Reid, Convention Dir., P. O. Box 456, Sunland Park, N.M. 88063. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MONTHLY OR WEEKLY

HEADACHE ROUNDS
Tuesday afternoons or Thursday mornings weekly. Sponsor: Headache Group of the Southwest. Contact: Peggy Voigt, 2610 W. Bethany Home Rd., Phoenix, AZ 85017, or phone 242-6646.

SHRINE MEDICS MEETING
Second Tuesday of each month, Doctors Hospital, 5:45 p.m., J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

OFFICE PSYCHIATRY FOR THE PRIMARY PROVIDER
2nd Monday of month, 4811 N 7th Street, Phoenix, AZ Sponsor Arizona Health Plan Contact T R Bittker, M.D., Box 5000, Phoenix, AZ 85010 Approved for required hours toward the ArMA Certificate in Continuing Medical

COUNTER TRANSFERENCE GROUP
Weekly, Thurs 8-10 p m Sponsor Phoenix Psychiatric Council Contact. James E Campbell, M D . 5051 N 34th St., Phoenix, AZ 85018. Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

MONTLHY MEETING OF TUCSON RADIOLOGISTS
Last Tues. of Month, Plaza International, Tucson, AZ. Sponsor U of A. Medical Center, Dept of Radiology Contact Irwin M. Freundlich, M.D., Arizona Medical Center, Dept of Radiology, Tucson, AZ 85724 Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

PEDIATRIC GRAND ROUNDS
Tuesday 7:30 - 8:30 a.m. in Phoenix: 1st Tue. - Phoenix Indian Hospital, 2nd Tue. - Maricopa County Hospital, 3rd Tue. - Good Samaritan Hospital, 4th Tue. - St. Joseph's Hospital
Sponsor: Phoenix Hospital's Affiliated Pediatric Program. Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

REVIEW OF FORENSIC PATHOLOGY, CURRENT CASE, SPECIAL TOPICS
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists, Contact: H.H. Karnit Schinig, M.D., 120 S. 6th Ave. Phoenix, AZ. Approved for 1 required hour per session toward ArMA CME Certificate.

Arizona State Hospital

STAFF EDUCATION CONFERENCE
Wednesdays, Weekly, 1 p m Arizona State Hospital, Phoenix, AZ. Sponsor Arizona State Hospital. Contact Howard E Wulsin, M D , Arizona State Hospital, 2500 E Van Buren, Phoenix, AZ 85008 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

Baptist Hospital

SURGICAL GRAND ROUNDS
4TH TUESDAY OF EACH MONTH
Hospital Auditorium, Baptist Hospital, Phoenix Sponsor Baptist Hospital Phoenix. Contact James B Shields, M D , 6036 N 19th Ave., Phoenix, AZ 85015. Approved for 1-1/2 required hours per month toward the ArMA Certificate in Continuing Medical Education.

Camelback Hospital

PATIENT STAFFING CONFERENCE
Three times weekly Camelback Hospital Phoenix, AZ Sponsor Camelback Hospital Contact. Medical Director, Camelback Hospital, 5055 N. 34th St., Phoenix, AZ 85018. Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

Good Samaritan Hospital

DESERT SAMARITAN HOSPITAL
Wednesday Evenings 7 p m. Sponsor Desert Samaritan Hospital Contact L A Rosati, M D., Laboratory, Desert Samaritan Hospital, Mesa, AZ 85202 Approved for required hours toward the ArMA Certificate in Continuing Medical Education

CLINICAL CANCER CONFERENCE
3rd Wednesday every month, Butler Bldg Conference Room, Good Samaritan Hospital, Phoenix, AZ. Sponsor Good Samaritan Hospital Contact John A Bruner, M D , 926 E. McDowell Road, Phoenix, AZ 85006 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

PULMONARY DISEASE GRAND ROUNDS
Mondays—12 Noon. D-5 North Conference Rm , Good Samaritan Hospital, Phoenix, AZ Sponsor Pulmonary Disease Teaching Service, Good Samaritan Hospital. Contact Bernard E Levine, M D , Pulmonary Function Laboratory, Good Samaritan Hospital, 1033 E McDowell Hospital, Phoenix, AZ 85006 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

Memorial Hospital, Phoenix

MONTHLY MEDICAL EDUCATION SEMINAR
Third Monday of the Month, 6 30 p m., Kiva Conference Room, Phoenix Memorial Hospital Sponsor Medical Staff of Memorial Hospital Contact George Scharf, M D , 1201 South 7th Avenue, Phoenix, AZ 85007 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

TUMOR BOARD CONFERENCE
Every Friday at Noon, Kiva Conference Room, Phoenix Memorial Hospital Sponsor Phoenix Memorial Hospital Contact Howard Kimball, M D , 333 West Thomas Road Phoenix, AZ 85013 Approved for credit toward the ArMA Certificate in Continuing Medical Education

Scottsdale Memorial Hospital

CLINICAL PATHOLOGICAL CONFERENCE
4th Monday, Monthly, 12 45 p m , Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor Medical Staff Contact R C Good, M D , Director of Medical Education, 7300 E 4th St., Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

FAMILY PRACTICE CONFERENCE
1st Monday, Monthly, 12 45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ. Sponsor Medical Staff Contact. R C. Good, M D., Dir of Medical Education, 7300 E. 4th St., Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

MEDICAL GRAND ROUNDS
3rd Monday, Monthly, 12 45 p.m., Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor: Medical Staff Contact R C Good, M D , Dir of Medical Education, 7300 E. 4th St., Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

MORBIDITY & MORALITY CONFERENCE
2nd Monday, Monthly, 12 45 p m , Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor Medical Staff Contact R C Good, M D , Dir Medical Education, 7300 E 4th St , Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

St. Mary's Hospital, Tucson

CARDIOLOGY CONFERENCE
Weekly—Friday 8-9 a.m., St Mary's Hospital Auditorium, Tucson, AZ Sponsor St Mary's Hospital Contact A L Forte, M D , St Mary's Hospital, Tucson, AZ 85724 Approved for one required hour toward the ArMA Certificate in Continuing Medical Education.

GRAND ROUNDS
Each Thursday 7 a m.-8 a m , St Mary's Hospital and Health Center, Sponsor Depts. of Medicine, Surgery, Radiology, Pathology and Family Practice. Contact Richard Silver, M D , Chairman, Medical Education and Library Committee, Century Medical Plaza, Suite 160, 1701 West St Mary's Road, Tucson, AZ 85703 Approved for 1 required hour per round toward the ArMA Certificate in Continuing Medical Education

MONTHLY SPECIALTY CONFERENCE OF THE DEPARTMENT OF SURGERY
First Monday, each month, 7:30 a.m., Century Room A. Sponsor: St. Mary's Hospital & Health Center. Contact: Medical Staff Office, St. Mary's Hospital & Health Center, P.O. Box 5386, Tucson, AZ 85703. Approved for one required hour per session toward the ArMA Certificate in Continuing Medical Education.

U of A Health Sciences Center

CLINICAL IMMUNOLOGY, ALLERGY AND RHEUMATOLOGY ROUNDS
Every Friday Noon-1 p m. Sponsor U of A College of Medicine, Dept of Internal Medicine, Clinical Immunology Section Contact John Boyer M D , U of A College of Medicine, Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

CEREBROVASCULAR DISEASE CONFERENCE
Mondays 5-6 p.m., weekly, Rm 5505 Surgery, U of AHSC. Sponsor: Depts. of Surgery, Neurology and Radiology, UAHSC. Contact: Dr. Wesley Moore, U of A Health Sciences Center, Tucson, AZ 85724.

ENDOCRINOLOGY SEMINAR
Every Thursday, Noon-1 p m , 1st, 3rd & 5th Thursday—Rm N318, VA Hospital, 2nd & 4th Thursday, Rm 6505, Tucson Medical Center. Sponsor U of A College of Medicine, Department of Internal Medicine, Department of Internal Medicine, Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

HEMATOLOGY-ONOCOLOGY CLINICAL CONFERENCE

Every Tuesday, Noon-1 p.m. 1st, 3rd & 5th Tuesdays – Rm 6505, AZ Medical Center 2nd & 4th Tuesdays – Rm. N318, Veterans Adm. Hospital. Sponsor: U. of A. College of Medicine, Dept of Internal Medicine. Contact Sidney Salmon, M.D., U. of A College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

MEDICAL GRAND ROUNDS

Every Wednesday, Noon-1 p.m. 1st, 3rd & 5th Wednesday – Staff Conf. Rm., VA Hospital 2nd & 4th Wednesday – Rm. 5403, Arizona Medical Center Sponsor U. of A College of Medicine, Dept. of Internal Medicine Contact Jay Smith, M.D., U of A College of Medicine, Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

NUCLEAR MEDICINE

Thursday, 7:30 a.m., Radiology Conf. Rm., AHSC, Tucson. Sponsor: Dept. of Diagnostic Radiology. Contact: Irwin M. Freundlich, U of A Health Sciences Center, Tucson, AZ 84724. Approved for one required hour per session toward ArMA CME Certificate.

NEUROLOGY GRAND ROUNDS

Every Friday, 12 noon. 1st, 3rd & 5th Fridays, U of A Health Sciences Center, Rm. 7505. 2nd and 4th Fridays, Veterans Administration Hospital. Sponsor: U of A Health Sciences Center, Dept. of Neurology. Contact: William A. Sibley, M.D., Dept. of Neurology U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

NEUROPATHOLOGY REVIEW CONFERENCE

Thursdays, 8:30 a.m., Rm 5120 Arizona Health Sciences Center. Sponsor: U of A College of Medicine. Contact: Peter C. Johnson, M.D., 1501 N. Campbell Ave., Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

NEUROSCIENCES SEMINAR

Every Friday, 7:30 a.m. U of A Health Sciences Center, Rm. 7505, Tucson, AZ. Sponsor: Dept. of Neurology. Contact: Colin R. Bamford, M.D. & Jose F. Laguna, M.D., Dept. of Neurology, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PATHOLOGY SEMINAR

Friday, 4:30-5:30 p.m., Rm 5120 AHSC. Tucson, AZ. Sponsor: Dept. of Pathology, Contact: Dr. Kenneth Ryan, Dept. of Path. U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour per session toward the ArMA Continuing Medical Education Certificate.

TUCSON PATHOLOGIST CONFERENCE

First Monday of the month, 7:30-9 p.m., Rotation Between Tucson Pathology Conf. Rms., Sponsor: Tucson Pathologists. Contact: Dr. Merle Clard, U of A Health Sciences Center, Dept. of Path., Tucson, AZ 84724. Approved for 1 required hour per session toward the ArMA Certificate in CME.

PEDIATRIC GRAND ROUNDS

Every Tuesday, 12:30 p.m. - 1:30 p.m. Alternates between Tucson Medical Clinic and U of A Health Sciences Center. Sponsor U of A College of Medicine, Dept. of Pediatrics. Contact: Hugh Thompson, M.D., 1501 N. Campbell, Tucson, AZ. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PROBLEM CASE WORKSHOPS

3rd Monday of each month 7:30 a.m. Room 4410, Arizona Medical Center, Tucson, AZ. Sponsor Division of Ophthalmology, U. of A College of Medicine. Contact H. E. Cross, M.D., Ph.D., Arizona Medical Center, Dept. of Surgery, Tucson, AZ Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

PSYCHIATRIC GRAND ROUNDS

Every Wed., Sept to May, 4-5:30 p.m. Rm. 8403, Arizona Medical Center, Tucson, AZ. Sponsor U. of A College of Medicine Dept. of Psychiatry Contact Alan Levenson, M.D., U of A College of Medicine, Tucson, AZ 85721 Approved for 1-1/2 required hour per session toward the ArMA Certificate in Continuing Medical Education

CHEST RADIOLOGY

Monday, 5-6 p.m., weekly, Rm. 1535F, AHSC, Tucson, AZ. Sponsor: Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of Radiology, Tucson, AZ 85724. Approved for 1 required hour per session toward ArME CME Certificate.

GASTROINTESTINAL RADIOLOGY

Mondays, 7:30 a.m., weekly except for summer months. Sponsor: Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., Diagnostic Radiology, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour toward ArMA CME Certificate.

RADIATION ONCOLOGY PLANNING CONFERENCE

Friday, weekly, 8:30-10 a.m., AHSC Rm. 0655. Sponsor: Radiation oncology Section, UAHSC, Tucson, AZ. Contact: Robert S. Heusinkveld, M.D., Ph.D., U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1.5 required hours per session toward ArMA Certificate in CME.

RADIOLOGY-RHEUMATOLOGY CONFERENCE

Thursday, 8 a.m., UAHSC, Library, Rm. 1535C. Sponsor: Dept. of Radiology, Contact: Irwin M. Freundlich, U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour toward the ArMA Certificate in CME.

VASCULAR RADIOLOGY

January thru June 1978. 7:30 a.m. to 8:30 a.m. every Friday. Dept. of Radiology. Sponsor: Dept. of Radiology, U of A Health Sciences Center. Contact: I. M. Freundlich, M.D., U of A Health Sciences Center Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

RADIOLOGY INTERESTING CASE CONFERENCE

Thursday, noon, Veterans Administration Hospital, Tucson, AZ. Sponsor: U of A Health Sciences Center, Tucson, Radiology Dept. Contact: Irwin M. Freundlich, M.D., U of A health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA CME Certificate.

RESIDENT'S CONFERENCE

Wednesday/weekly 5-6 p.m., Diag. Radiology Conf. Rm., U of A Health Sciences Center. Sponsor: U of A Health Sciences Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour per session toward the ArMA Certificate in Continuing Medical Education.

TRAUMA CONFERENCE

Every Thursday, 4-5:00 p.m. Health Sciences Center, Rm. 5505, Tucson, AZ. Sponsor: U of A College of Medicine, Dept. of Surgery. Contact: Martin Silverstein, M.D., Dept. of Surgery, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

GRAND WARD ROUNDS – TRAUMA

Every Tuesday, 8 a.m. Arizona Medical Center, Tucson, AZ Sponsor U. of A. College of Medicine, Surgery Dept., Trauma Section Contact Martin Silverstein, M.D., U of A. College of Medicine, Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

MONTHLY MEETING TUCSON ULTRASONOGRAPHY GROUP

First Wednesday of month, 4:30-5:30 p.m., Arizona Health Sciences Center. Sponsor: Dept. of Diagnostic Radiology. Contact: Irwin M. Freundlich, M.D., AHSC, Tucson, AZ 85724. Approved for 1 required hour per session toward ArMA CME Certificate.

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87th Annual Meeting in '78

Plan to attend the 1978 Annual Meeting April 25-29, 1978 at the Tucson Community Center and Marriott Hotel —

Mark your calendar.

TUCSON UROLOGICAL SOCIETY SCIENTIFIC MEETING
Monthly, Sponsor: Depts of Surgery & Urology, AHSC, Tucson. Contact: George W. Drach, M.D., U of A ealth Sciences Center, Tucson, AZ 85724. Approved for 1.5 required hours per session toward ArMA CME Certificate.

GENERAL UROLOGY CONFERENCE
Tuesday 5-6 p.m., Thursday noon-1:30 p.m. AHSC Meeting rms., & VA Hospital. Sponsor: Dept of Surgery/Urology Section. Contact: George W. Drach, M.D. Dept. of Surgery/Urology Section, U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour toward ArMA CME Certificate.

VA Hospital, Phoenix

CANCER SYMPOSIUM (fomerly Tumor Board)
Each Wednesday, 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact. Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

CARDIOLOGY CONFERENCE
2nd Thursday 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact Jasper L McPhail M.D., VA Hospital, 7th & Indian School Rd Phoenix, AZ Approved for required hours toward the ArMA Certificate in Continuing Medical Education

MEDICAL-SURGICAL CHEST CONFERENCE
1st and 3rd Thursday, 1 p.m., T-5 Sponsor VA Hospital Phoenix. Contact. Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

CLINICOPATHOLOGY CONFERENCE
4th Thursday of 3rd Mo (Mar., June., Sept & Dec.), 1 p.m., T-5 Sponsor VA Hospital Phoenix. Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ Approved for required hours toward the ArMA Certificate in Continuing Education.

DERMATOLOGY CONFERENCE
1st, 2nd & 3rd Wednesday Sponsor VA Hospital Phoenix Contact. Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

HEPATOLOGY CONFERENCE
2nd and 4th Tuesday, 1 p.m., 2128 Sponsor VA Hospital Phoenix. Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

GASTROENTEROLOGY CONFERENCE
1st and 3rd Tuesday, 1 p.m., T-5 Sponsor VA Hospital Phoenix, Contact. Jasper L McPhail, M.D. VA Hospital, 7th & Indian School Rd., Phoenix, AZ Approved for required hours toward the ArMA Certificate in Continuing Medical Education

GI CONFERENCE
(Special Program with U of A Consultants) 4th Friday, 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

G.I.-RADIOLOGY CLINICAL CORRELATION CONFERENCE
Each Tuesday, 1 p.m., T-5. Sponsor: VA Hospital Phoenix. Contact: Jasper L. McPhail, M.D., Veterans Administration Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL SURGICAL G.I. CONFERENCE
1st & 3rd Friday - 2 p.m. -2128 Sponsor: VA Hospital, Phoenix. Contact: Jasper L. McPhail, M.D., VA Hospital, 7th Ave. & Indian School Rd., Phoenix. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL SERVICE GRAND ROUNDS
Each Friday, 11 a.m., T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

SURGERY-PATHOLOGY CONFERENCE
Each Thursday, 7 a.m., 2128 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

UROLOGY-PATHOLOGY CONFERENCE
Each Wednesday, 7 a.m., 2128. Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th and Indian School Rd., Phoenix, AZ Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

AMA Law Department Provides Information and Assistance on Request...

The Health Law Department of the Office of the General Counsel reviews, complies and evaluates legal information on topics which affect the physician in his medical practice. It provides information and assistance to physicians and their attorneys on request.

Examples of areas within the scope of this Department's activities include medical malpractice, hospital-physician relations (including medical staff bylaws review and staff privileges concerns), allied medical professionals, legal aspects of medical office practice, and government regulations.

Attorneys in this department provide legal support for a number of AMA activities, such as the AMA's practice management workshops, medical and allied health education programs, professional liability task force and the project on health care in jails. This department also provides legal support for various state and local medical society programs.

AMA Department of Health Law
535 North Dearborn Street
Chicago, Illinois 60610

For additional information, write to Donald P. Wilcox, J.D., Director, at the above address or telephone: (312) 751-6178.

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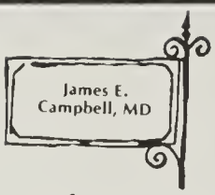
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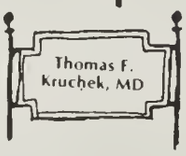
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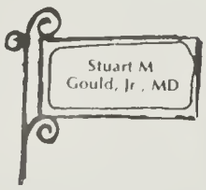




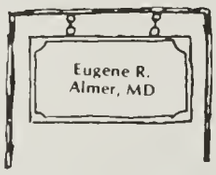


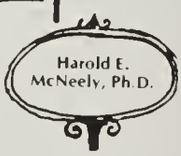












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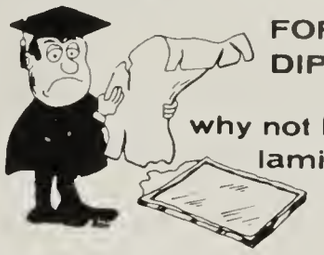
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Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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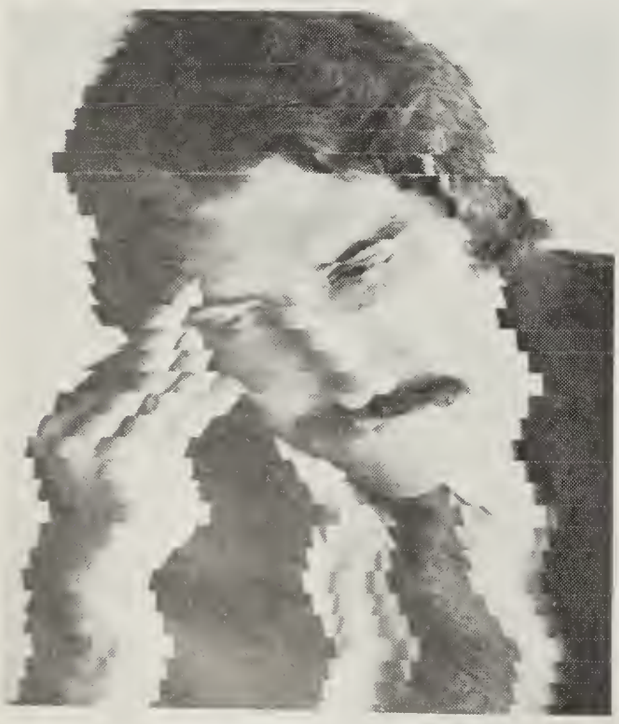
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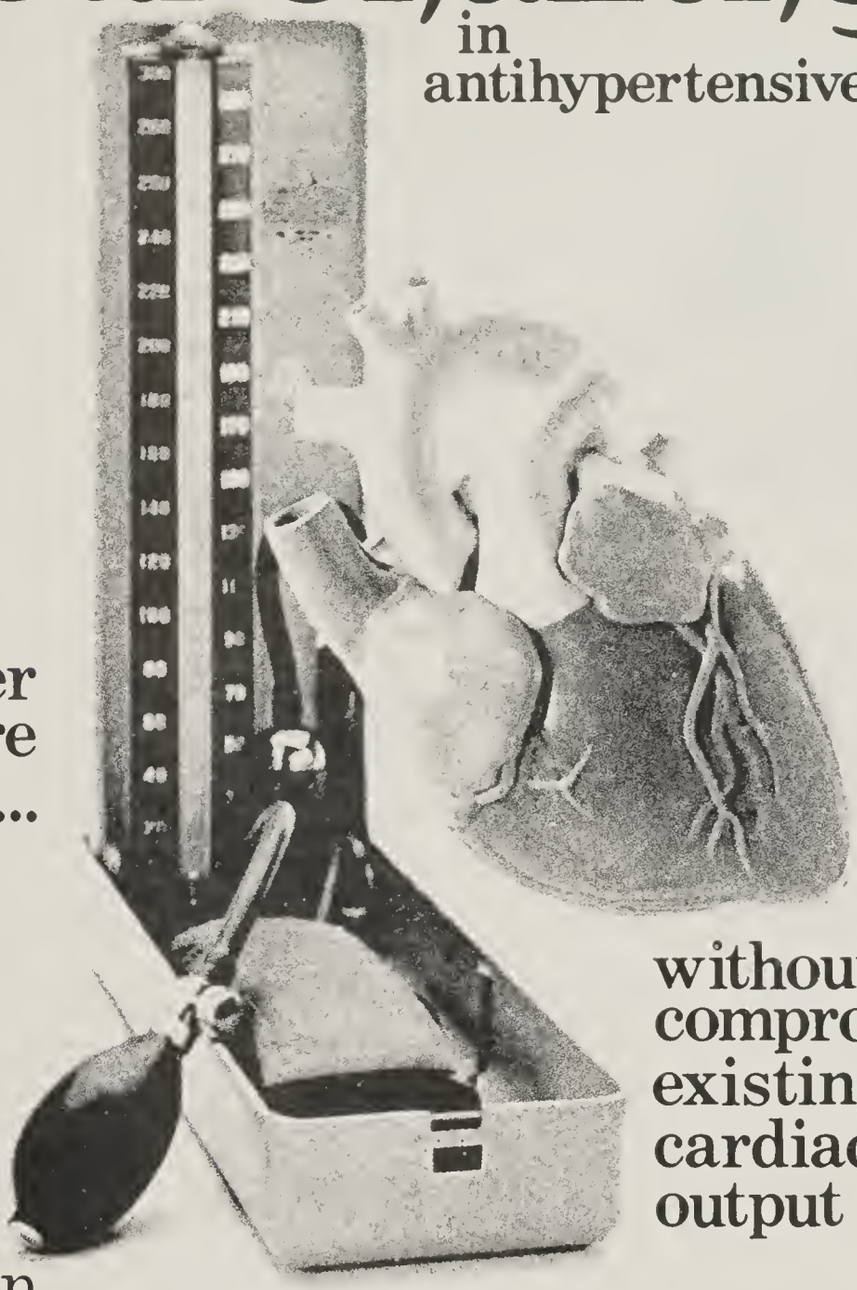
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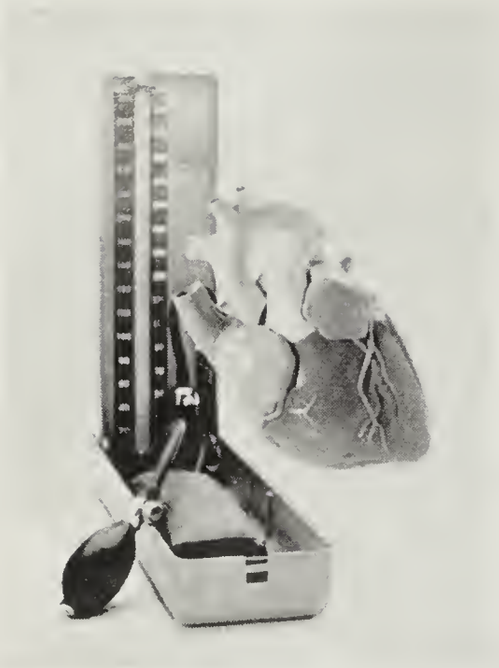
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With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood.

At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstituted. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, occasionally with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstituted in such patients.

Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

Pregnancy and Nursing: Use of any drug in women who are or may become pregnant or intend to nurse requires that anticipated benefits be weighed against possible risks; possibility of fetal injury or injury to a nursing infant cannot be excluded. Methyldopa crosses the placental barrier, appears in cord blood, and appears in breast milk.

Precautions: Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: urinary uric acid by the phosphotungstate method, serum creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetotic movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyldopa because the drug is removed by this procedure.

Adverse Reactions: *Central nervous system:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetotic movements; psychic disturbances, including nightmares and reversible mild psychoses or depression.

Cardiovascular: Bradycardia, aggravation of angina pectoris. Orthostatic hypotension (decrease daily dosage). Edema (and weight gain) usually relieved by use of a diuretic. (Discontinue methyldopa if edema progresses or signs of heart failure appear.)

Gastrointestinal: Nausea, vomiting, distention, constipation, flatus, diarrhea, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis.

Hepatic: Abnormal liver function tests, jaundice, liver disorders.

Hematologic: Positive Coombs test, hemolytic anemia. Leukopenia, granulocytopenia, thrombocytopenia. Positive tests for antinuclear antibody, LE cells, and rheumatoid factor.

Allergic: Drug-related fever, lupus-like syndrome, myocarditis.

Other: Nasal stuffiness, rise in BUN, breast enlargement, gynecomastia, lactation, impotence, decreased libido, dermatologic reactions including eczema and lichenoid eruptions, mild arthralgia, myalgia.

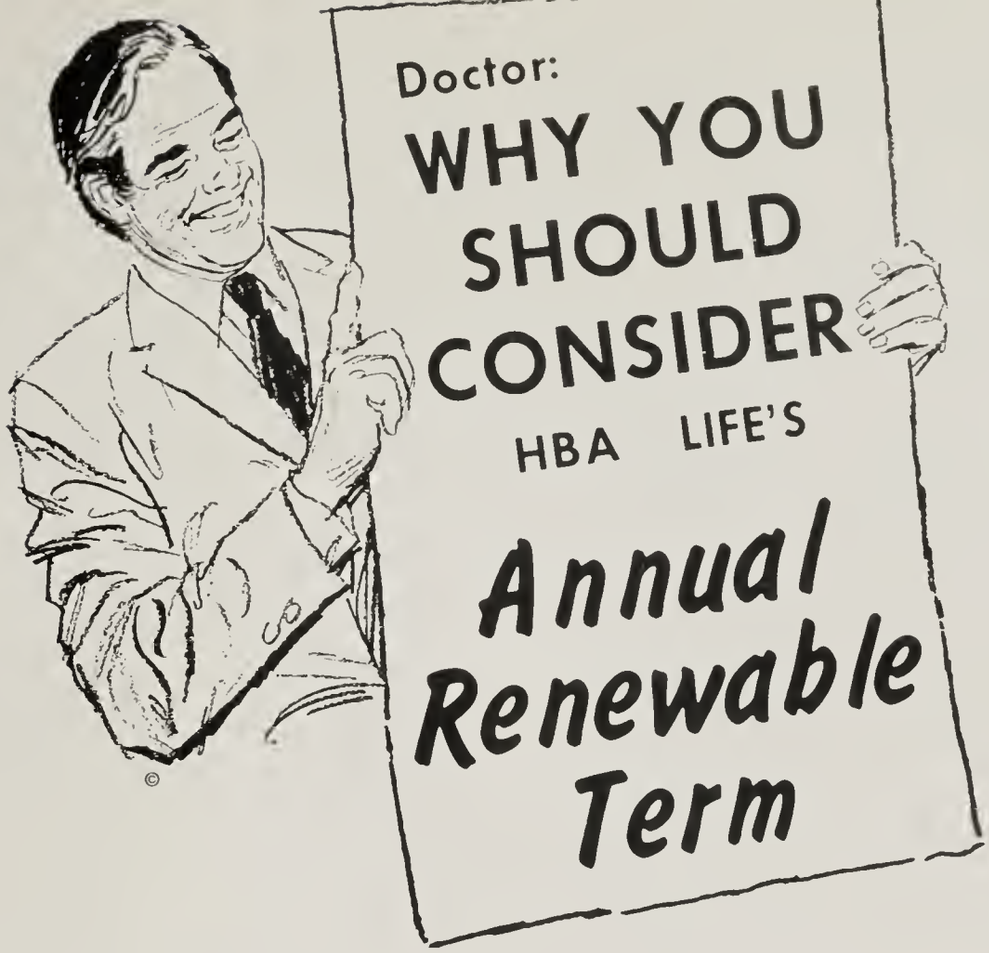
Note: Initial adult dosage should be limited to 500 mg daily when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third months of therapy; increased dosage or adding a diuretic frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

How Supplied: Tablets, containing 125 mg methyldopa each, in bottles of 100; Tablets, containing 250 mg methyldopa each, in single-unit packages of 100 and bottles of 100 and 1000; Tablets, containing 500 mg methyldopa each, in single-unit packages of 100 and bottles of 100 and 500.

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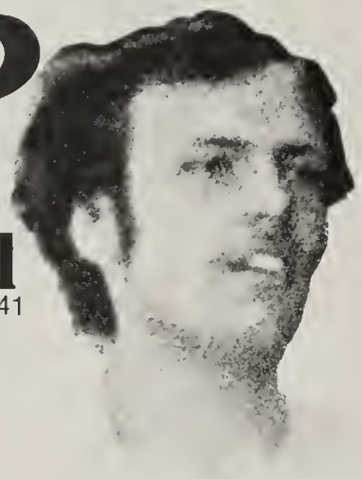
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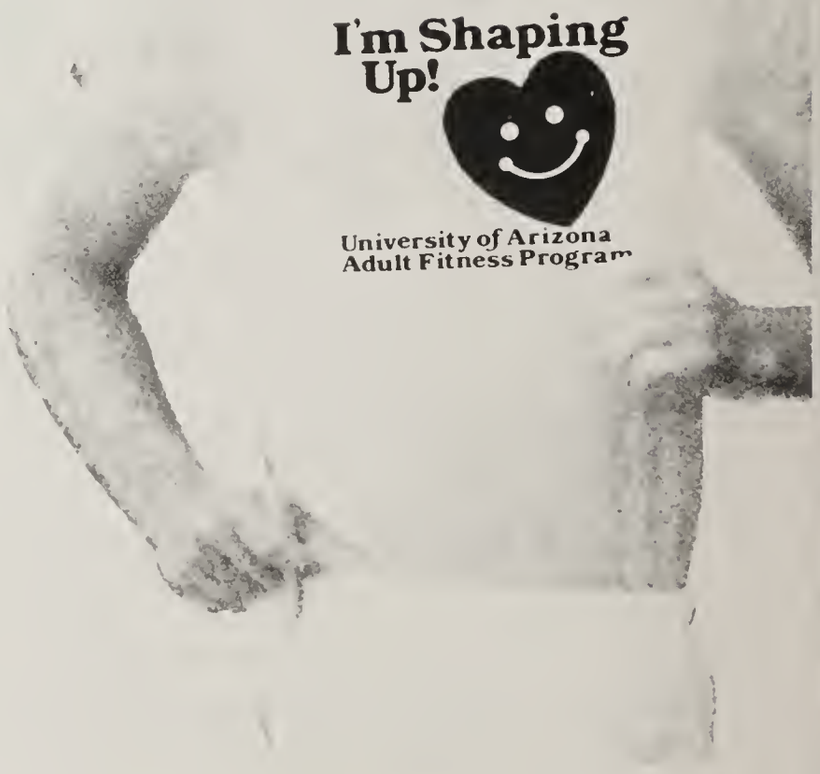


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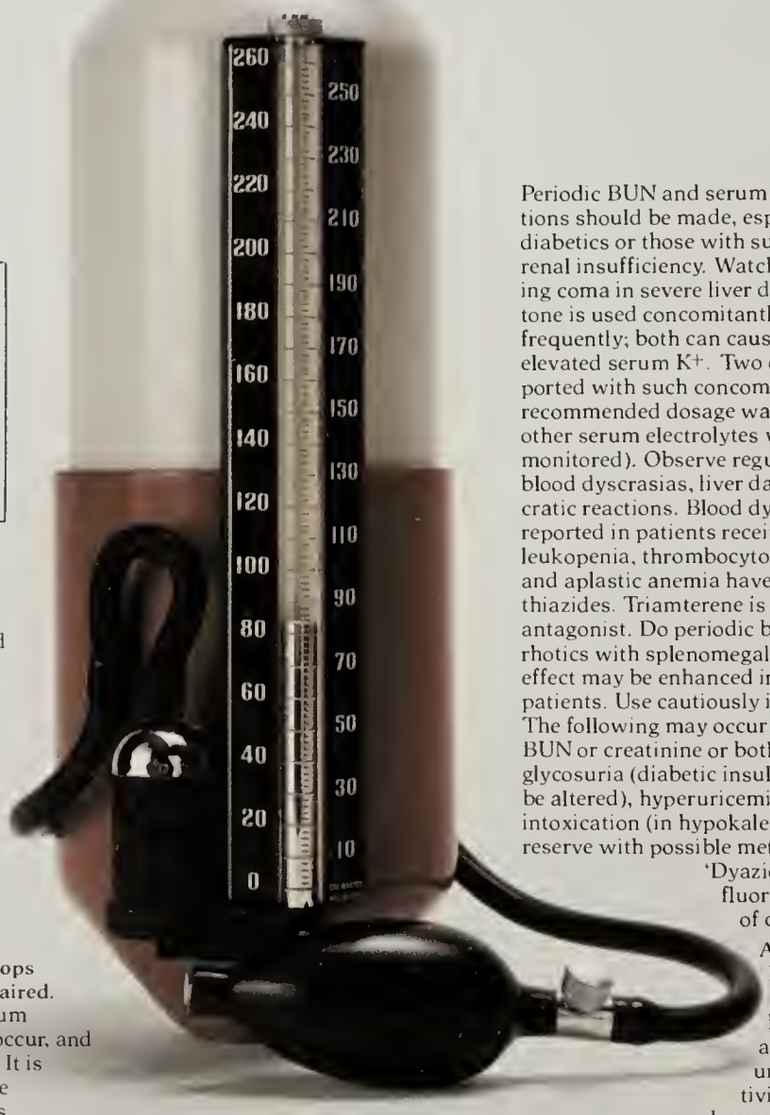
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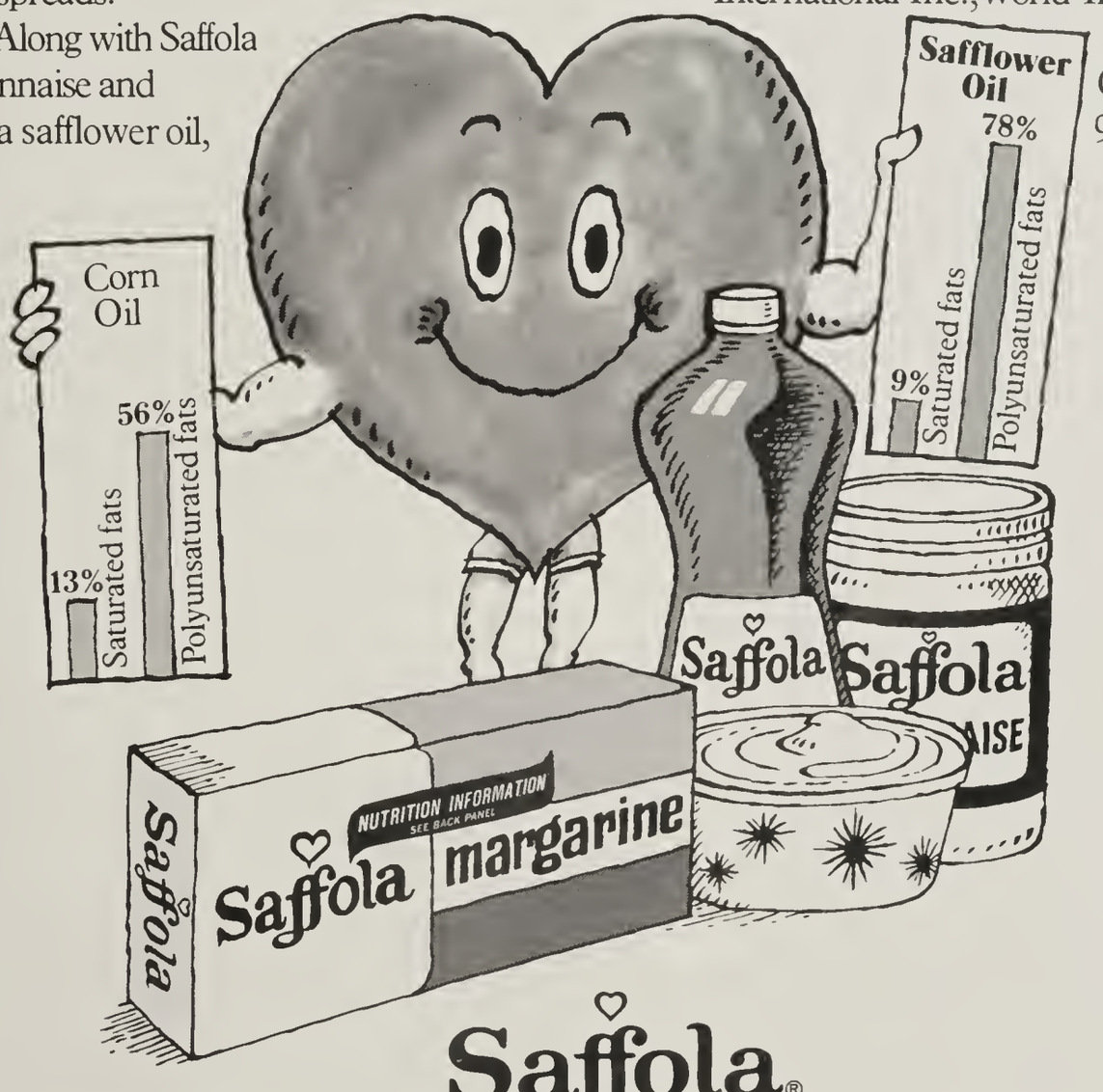
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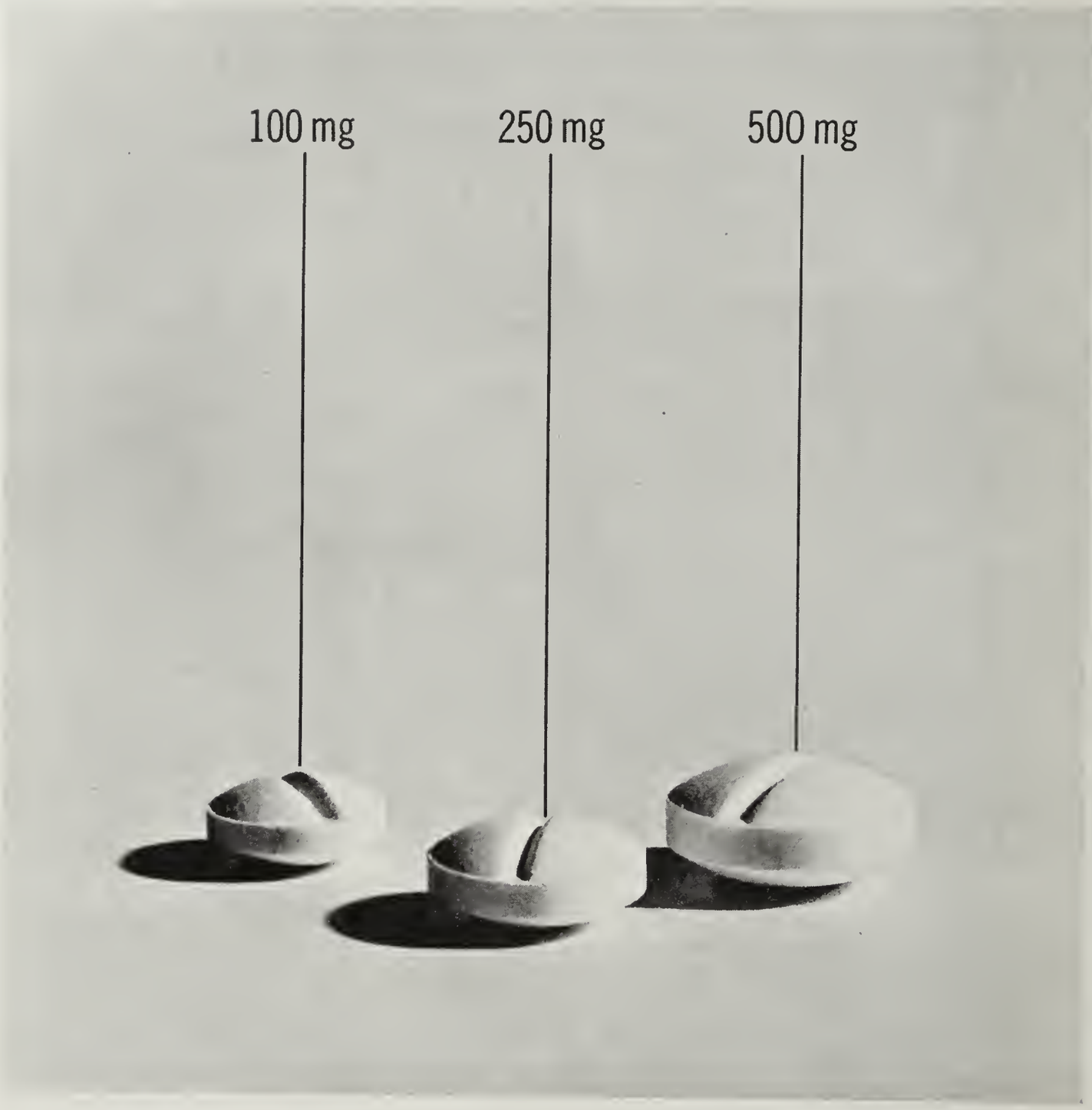
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Seminars in Chest Medicine

Current Status Of The Chemotherapy Of Tuberculosis

Part II

Gregory L. Lauver, M.D.

Editors:
Robert J. Clark, M.D.
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Pharmacology of First Line Drugs

Isoniazid. INH is administered in a dose of 5-10 mg/kg/day, often given as a single dose of 300 mg in the morning. The dose is bacteriocidal to the tubercle bacillus through inhibition of nucleic acid synthesis. It is rapidly absorbed from the gastrointestinal tract, metabolized by hepatic microsomal enzymes, and excreted in the urine. INH is well distributed into body fluids including cerebrospinal fluid and caseous material. Side effects include peripheral neuritis which is preventable with pyridoxine, hypersensitivity reactions, lupus erythematosis-like reactions, and chemical hepatitis. Before starting INH, liver function tests should be obtained. Hepatic toxicity of INH will be discussed later in the section on chemoprophylaxis.

Ethambutol. Ethambutol is given orally in a dose of 15 mg/kg/day (usually resulting in a dosage of 800-1200 mg daily). In severe disease, the initial dose may go as high as 25 mg/kg/day for up to 60 days. The necessity for higher doses however, must be weighed against the approximate 10% incidence of occular toxicity associated with the 25 mg/kg dose. The drug acts by inhibition of RNA synthesis. It is about 75-80% absorbed from the gastrointestinal tract, metabolized in the liver, and excreted in the urine. Optic neuritis with loss of green-red color discrimination is the most common toxicity; this is reversable by discontinuing the drug. An initial funduscopic exam, visual acuity, and color vision examination are required prior to therapy, and follow-up color vision testing is recommended.

From: Division of Respiratory Sciences, University of Arizona Health Sciences Center, Tucson, AZ 85724. Dr. Lauver, Parker B. Francis Fellow in Pulmonary Diseases.

FIGURE 1
ADVERSE REACTIONS WITH INTERMITTENT RIFAMPIN THERAPY

1. Cutaneous reactions, flushing, pruritis, rash
2. Facial redness and watering of eyes
3. Abdominal reactions; with pain, vomiting, and diarrhea
4. "Flu-like" syndrome—myalgia, fever, malaise
5. Respiratory reactions with dyspnea and wheezing
6. Thrombocytopenia, Purpura

Rifampin. Rifampin, an inhibitor of RNA polymerase activity, is usually administered as a once daily oral dose of 600 mg. It is well absorbed from the intestinal tract, metabolized by liver enzymes, and excreted in the feces. A variety of serious toxic reactions and unusual laboratory findings have been reported with rifampin. The most curious of those associated with daily therapy are the qualitative changes in immunoglobulin polymerization with resulting light-chain proteinuria. Rifampin has been reported to inhibit cell-mediated rosette formation *in-vitro*. Adverse reactions associated with intermittent rifampin administration are listed in Figure 1. The most severe among these are the rare hematologic syndromes resulting in hemolysis, thrombocytopenia, renal failure and death. Accordingly, rifampin is not administered in intermittent therapy programs. An incidental note is that rifampin colors the urine orange.

Streptomycin. Streptomycin is an injectable antibiotic of the aminoglycoside type which is given in a dose of 0.75-1.00 grams intramuscularly daily as a single injection. A total dose of 60-120 grams given over two to

four months is usually satisfactory. Streptomycin binds to the tubercle bacillus ribosome and disrupts RNA synthesis. The drug is well distributed into caseous material in cavities, but its penetration into cerebrospinal fluid is poor. It is excreted in the urine. Major toxicity from streptomycin is vestibular (vertigo) and auditory (hearing loss and tinnitus). Renal dysfunction is commonly observed with higher doses. Routine audiograms should be performed on patients receiving Streptomycin.

PAS. Para-aminosalicylic acid is chemically similar to aspirin and is administered in a dose of 12-15 grams daily. Its major drawbacks are the severe gastrointestinal side effects of nausea and vomiting caused by large doses of this aspirin-like chemical. Accordingly, patient compliance is poor and the drug is rarely employed in initial therapy at present.

Preventive Therapy for Tuberculosis

Two forms of preventive therapy for tuberculosis are currently employed in the United States. The first of these, BCG-vaccination, is the intradermal injection of a non-virulent strain (Bacillus-Calumette-Guerrin) for the actual *prevention* of infection before it occurs. This approach has been used in Africa, Asia and to a lesser degree, in larger American cities. BCG converts the PPD skin test permanently to positive and thus eliminates the use of the PPD as a screening device in BCG-vaccinated populations.

The second, and more widely used method, INH-chemoprophylaxis, is actual *therapy* for an infection already present but not clinically manifest. The remainder of this section will deal with INH chemoprophylaxis and some of the special considerations associated with INH.

FIGURE 2
INH CHEMOPROPHYLAXIS IN TB PREVENTION
"Which Patients Should be Treated"

1. Recent PPD convertors (especially under age 35)
2. Household contacts and close associates of new active and reactivated cases of TB
3. Persons with inactive TB who:
 a) have positive PPD, compatible chest x-ray, negative sputum
 b) previously known TB but who received no prior chemotherapy
4. All children with positive PPD
5. Special circumstances

INH Chemoprophylaxis

The American Thoracic Society has adopted guidelines for the use of INH in the prevention of tuberculosis.[15,16] These recommendations are summarized in Figure 2. Essentially they revolve around a history of exposure to an active case of tuberculosis and/or the presence of a positive PPD skin test. Special circumstances are also recognized including: 1) prolonged steroid or immunosuppressive therapy; 2) after-gastrectomy; 3) with malignancies; 4) in poorly controlled diabetes; and 5) with evidence of silicosis. The institution of INH chemoprophylaxis is not an automatic event once a recent PPD convertor or contact has been identified. Instead, the risk of developing tuberculosis, age considerations, and finally potential drug toxicity are all carefully weighed, and a decision on whether to start therapy is reached.

The possibility of developing active tuberculosis for any population at risk has been estimated.[1] Recent convertors have a 3% risk of active disease in the first year and a 3-5% risk over the next five years. Abnormalities in the chest roentgenogram increase the risk such that over a lifetime, persons with a positive PPD and an abnormal chest roentgenogram have a tenfold greater chance of developing tuberculosis than those with a normal roentgenogram. Since clinicians rely on a positive PPD for initiation of INH therapy, they should be aware of certain situations in which latent or active infection may be present but the tuberculin skin test is negative. The causes of a false-negative PPD are listed in Figure 3.

FIGURE 3
FALSE NEGATIVE TUBERCULIN SKIN TEST

1. Non-tween-80 stabilized product
2. Old or outdated test material
3. Sub-Q rather than intradermal injection
4. Recent vaccination or infection with: rubella, polio, rubeola
5. Immunosuppressive with steroids or cytotoxic agents
6. Fulminant TB
7. Severe illness, malnutrition, dehydration
8. Advanced age
9. Lymphoreticular malignancies: Hodgkins and Lymphoma

The toxicity of INH has been well described and is primarily that of chemical hepatitis.[18,19] INH hepatotoxicity occurs in about 1% of the recipients of the drug. Transaminase enzymes (SGOT, SGPT) are elevated in about 10% of the recipients, but most of these patients are asymptomatic and unaware of the "transaminitis." Such liver toxicity is more common in patients older than 35-40 years of age. Liver injury is more likely to occur within the first two months of therapy. Susceptability to liver damage is probably genetically determined by the type of acetylating enzyme present in the liver. Rapid hepatic acetylators are at higher risk than slow acetylators because rapid metabolizers generate large amounts of hepatotoxic metabolites quickly and thereby sustain more severe liver injury. Acetylhydrazine is one such hepatotoxic metabolic product which accumulates. Elevations of SGOT of less than 2.5 times normal are not serious and therapy can be continued. Higher elevations of SGOT, the presence of a flu-like illness, jaundice, or eosinophilia are felt to be harbingers of more serious INH reactions, and the drug must be stopped.

Cost-benefit studies have been done calculating the number of cases of tuberculosis preventable by INH prophylaxis and the number of cases of INH hepatitis.[18] From these studies cost-benefit ratios in the form of numbers of cases of TB prevented per case of INH

Table I
RATIO OF T.B. CASES PREVENTABLE
BY INH TO HEPATITIS CASES

Race	Sex	Age Years 25	35	45	55	65
W	M	23.7	4.2	1.9	0.9	0.8
W	F	19.5	3.7	1.8	1.0	1.1
B	M	25.8	3.9	1.7	0.8	0.8
B	F	24.7	4.3	1.8	0.9	1.0

Ratio:TB Prevented/Definite and Probable Hepatitis

hepatitis caused can be derived (Table I). For patients under age 35 there is a distinct case-ratio advantage in prescribing INH prophylaxis (e.g., for Caucasian males at age 25, almost 24 cases of tuberculosis are prevented for every case of hepatitis observed; similarly at age 35, the ratio is 4.2). There is a gray zone in the age range 45-55 where the ratios are closer to unity. Beyond age 55 there is a definite statistical hazard to giving INH. The risk of hepatitis must be seriously weighed against the risk of tuberculosis before initiating therapy in the older age groups.

FIGURE 4
CRITERIA FOR DISCHARGE OF
TB PATIENT FROM MEDICAL SUPERVISION

1. Adequate chemotherapy
 a) 2 years of multiple drug therapy or
 b) 1 year of multiple drugs plus 1 year of INH
2. Resolution of pulmonary and systemic symptoms
3. Resolution or stabilization for 1-2 years of chest x-ray
4. Continued negative sputum cultures
5. Patient educated to return if pulmonary and/or systemic symptoms occur

Finally, once adequate chemotherapy has been delivered a question of follow-up arises. Many physicians place former tuberculosis patients on indefinite annual follow up. The validity of this practice has been drawn into question.[20] Several guidelines for discharge of a tuberculosis patient from medical supervision are available[1] and are summarized in Figure 4. They are all based on the standard 18-24 month chemotherapy. These standards may change as short-course chemotherapy gains acceptance.

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Dental Disease

Periodontal (Gum) Disease In Adolescents

Perry A. Ratcliff, D.D.S., M.S.

Puberty is frequently accompanied by an exaggerated response of the gingiva to local irritants.

Common causes of periodontal irritation in adolescents are due to the failure of the teenager to maintain adequate oral hygiene. There is a lack of adequate removal of bacterial dental plaque, removal of calculus deposits, presence of mouth breathing, presence of untreated carious lesions, and the complicating effects of occlusal disharmony resulting in food impaction and irritation. Dental plaque forms more rapidly with the higher sucrose diet common with many teenagers. Mouth breathing with dehydration of the mucosa in the maxillary anterior portion of the mouth cause an increase in the prevalence of gingival hyperplasia. Crowding of the anterior teeth by malocclusion aggrevates the problem.

Although the prevalence and severity of gingival disease is increased in puberty, not all adolescent patients have pubertal gingivitis. Discrimination must be made between other conditions which can present similarly.

From: 7125 E. Lincoln Dr., Scottsdale, AZ 85253.

Dilantin hyperplasia is a response of the gingiva to the metabolites of the drug diphenylhydontoin sodium. This drug is commonly used in the treatment of epilepsy and occasionally with other neuropathies. There is a marginal and interdental proliferative lesion aggravated by local oral irritation, without appreciable extension into the adjacent attached gingiva or oral mucosa. When uncomplicated by irritation the clinical appearance is of a pink, stippled, fibrous overgrowth of gingiva. Histologically it is characterized by engorged prominent cappillaries and numerous proliferating fibroblasts in a meshwork of collagen strands. Differential diagnosis is made by a history of drug intake and location of the hyperplasia.

Fibromatosis is also known as hereditary or familial fibromatosis, idopathic fibrous hyperplasia, diffuse gingival fibroma and gingivomatosis elephantiasis. It involves the marginal, interdental and attached gingival tissues in contrast to Dilantin hyperplasia which involves only the marginal and interdental gingiva. Local irritants may complicate the initial lesion by superimposition of inflammation. Clinical appearance is often very similar and differentiation is best determined by a history of drug intake. In the absence of Dilantin use the clinician should seek out a possible history of similar manifestations by parents or siblings.

Leukemia is expressed as enlarged gingival tissues by an exaggerated response to the irritation of toxins from oral microorganisms. Variations in clinical appearance depend on the degree of leukocyte incompetence and the level of oral hygiene. Not all patients with leukemia will have gingival changes. Histologically the cappillaries may be engorged and the connective tissue edamatous and degenerated. This is in contrast to the fibrous character of pubertal gingivitis, Dilantin hyperplasia and fibromatoisis.

Vitamin C deficiency, a rare disease in modern U.S. culture, does not in itself cause gingival inflammation or enlargement. When scorbutic gingivitis is present it is characterized by hemorrhage, presence of tropocollagen rather than mature collagen and edema as in other body tissues when irritation is added to the primary scorbutic lesion. The inflammed gingiva is blueish red, soft and friable with a smooth surface. The clinical appearance is more reminiscent of leukemia than the fibrous lesions of pubertal gingivitis, Dilantin hyperplasia or fibromatoisis.

Pregnancy may be complicated by localized gingival enlargement as well as a generalized gingivitis of pregnancy. Not all pregnant women have gingival manifestations. Those that show generalized changes usually are characterized by edema, magenta coloration, glossy tissue surface and bleeding with gentle probing. Occasionally there will be a fibrotic appearance. Gingival tumors of pregnancy usually appear as single or multiple papillary enlargements, most often at an interdental location. Differential diagnosis is by determination of pregnancy and eliminating white cell dyscrasias and scorbutic changes. There is rarely the generalized fibrotic enlargements of pubertal gingivitis, Dilantin hyperplasia or fibromatoisis. There may be a similarity to a localized periodontitis.

Inflammatory hyperplasia caused entirely from local irritation is a common finding in the adult and a portion of the adolescent population. It is the inflammatory fibrous hyperplastic response to bacterial plaque, calculus deposits, ill fitting crowns and fillings, untreated carious lesions and other local irritations of the mouth. The lesion starts as an interdental or marginal gingivitis and becomes a periodontitis as the deeper attachment apparatus of the tooth becomes involved. The layman knows this process as "pyhorrea". Epidemiology studies show the disease incidence and prevalance to parallel that of arthrosclerosis. The World Health Organization lists the disease as man's most prevalant malady. Differential diagnosis is made by the identification of the presence of local irritants and ruling out of the above similar clinical phenomena.

Treatment is to identify all local irritations present and remove them as etiologic agents. When extensive hyperplasia of the tissues has brought about permanent architectural deviations from normal, a gingivoplasty may be required to make hygiene easier and improve cosmetics. If the tissues have had only an edemoutous response the architecture will usually assume approximately that of physiologic form after the irritants have been removed. The most difficult problem is to get the pubertal aged patient to institute an adequate home care regime to keep bacterial plaque off the teeth and the marginal gingiva.

Since periodontal disease can be the harbinger of more serious dental problems later in life its recognition early, in the adolescent, may help prevent significant morbidity in adulthood. It would seem prudent to pay more attention to the mouth during routine physical examinations.

Drug Therapy Problems

Oral Morphine For Control of Pain In Terminal Cancer

William C. Farr, Ph.D., M.D.

ABSTRACT

The pain of terminal cancer is treatable by a number of modalities including analgesics. The majority of patients can be maintained pain-free and alert until death on oral medications used appropriately. Treatment of cancer pain should begin with non-narcotics and should remain thus until the severity of pain increases. Narcotic analgesics can be added without fear of producing psychological dependence or development of significant tolerance. Chronic pain, unlike acute postoperative or traumatic pain, is complicated by psycho-social-religious components that need to be treated individually. With moderately severe chronic pain, the routine prescription of an analgesic on a p.r.n. basis is less effective than a preventive schedule. In addition to the many oral morphine-like drugs that are commercially available, an elixir of morphine (modified Brompton mixture) is effective and has many advantages. Oral morphine is as effective as parenteral morphine in the control of the physical pain of terminal cancer if used properly and in high enough doses.

Dying with chronic, unrelenting pain remains one of the greatest fears of the terminally ill cancer patient. The pain of terminal cancer is more than just a physical pain; it is emotional, social, economic, and religious. Each aspect must be recognized and handled differently but all must be considered if the patient and his family are to live fully this "last stage of life." In England, and now in many countries including the United States, the hospice with its multidisciplinary team approach to the terminal patient is proving to be a logical answer to the pains of terminal illness. With years of experience behind them, the British hospice programs have consistently found the use of "The Brompton Mixture" a practical and effective analgesic for use in managing the physical pain of terminal cancer. The purpose of this paper is to review some of the principles of treatment of physical pain using the modified Brompton mixture. The methods used are particularly applicable to the treatment of the outpatient, and thus have considerable significance to the practitioner for use in his home care patients.

With the armamentarium available to the physician today, including chemotherapy, radiation therapy, neurosurgical techniques, and a wide range of analgesic medications, it seems appropriate that the physician, in the early stages of cancer diagnosis, reassure his patient that severe pain is not always an associate of a malignancy. The reassurance early in the disease can be of great comfort to the patient and his family. It is the lack of success in treating the pain of terminal cancer that permeates the lay literature, and not the successes. Statistics show that about 50% of cancer patients have little or no physical pain during their illness. Another 10% have only mild pain and of the remaining 40%, 50% of these will need help with intractable pain.[1] A majority of the latter groups can be adequately controlled by a number of effective palliative techniques including analgesics. Statistics from St. Joseph's Hospice in England show that 85% of the terminally ill cancer patients are comfortable until death[2] with a similar percentage of patients maintained pain-free and alert using only oral medications.[3]

The fact that oral analgesics can be as effective as the parenterally administered drug in the treatment of severe oncologic pain is not generally appreciated by the practitioner. It is important to use an oral medication before parenterally administering analgesics. The reasons are simple. The injection of a narcotic produces an unnecessary anxiety reaction in the patient. We hear statements from patients such as "Now that injections (narcotics) are necessary, what if it doesn't work?" Also, the injection is not without discomfort. Most importantly, repeated intramuscular or subcutaneous injections over a prolonged period of time in a debilitated patient can lead to skin breakdown and increased discomfort. Finally, injectables are more expensive than oral medication and, for administration, require a skilled person, or the training of a family member.

From: Hillhaven Hospice, (A not-for-profit Corp.) 5504 E. Pima St., Tucson, AZ 85712. (Dr. Farr, Ph.D., M.D., Director).

TABLE I
EQUIANALGESIC DOSES OF COMMONLY USED DRUGS

Generic Name	Trade Name	Dosage (mg)	Duration (hour)	Oral-parenteral ratio
Morphine		10	4-5	1/2-1/6 low
Hydro-morphone	Dilaudid	2	3-4	1/4 low
Meperidine	Demerol	80-100	2-4	1/3-1/4
Levorphanol	Levo-Dromoran	2-3	4-5	1/2 high
Oxycodone	Percodan	10-15	4-5	1/2 high
Codeine		120	4-5	1/2 high

THE PAIN SYNDROME:

Table 1 lists the most commonly used analgesics in equally effective doses. The selection of a pain control program is based upon the severity of the pain and the patient's psychological reaction to it. Unlike the acute pain secondary to surgery or injury, both of which are expected by the patient to be of short duration, chronic pain of terminal illness is significantly influenced by the patient's emotional reaction to the recurrent nature of his discomfort. Attention to the patient's psychological state, be it anxious or depressed, can be extremely helpful. Selection of a psychotropic drug may have profound effects on the dose of analgesic needed. For instance, the treatment of depression or anxiety may lower the dose of analgesic necessary to maintain the patient in comfort and reduce the incidence of untoward side effects of the analgesic.[4]

THE PREVENTIVE SCHEDULE:

Timing of the administration of an analgesic can be of utmost importance in the treatment of cancer pain. If the patient's pain is intermittent and of short duration, the use of analgesics as needed (on a p.r.n. basis) is appropriate. However, when the pain becomes chronic and unrelenting, the institution of a preventive analgesic schedule is quite effective.[3,4] At St. Christopher's Hospice in England and now in many other hospices, analgesics are administered on a regular schedule based on the duration of action of the drug. The medication is given around the clock, whether the patient is in pain or not. A regular schedule utilizes the benefits of cumulative dose effect, thus establishing a blood and tissue level of analgesic that is smooth and effective. This principle of cumulative dose is quite similar in concept to the pharmokinetic basis of the use of maintenance doses of digitalis. The maintenance dose of an analgesic is usually lower than when the drug is used on an as needed basis. Consequently, the wide swings in blood levels of analgesics, with their attendant side effects on the mental status and respiration, can be avoided. There is a psychologically positive "spin-off" of establishing an effective preventive protocol. It reduces patient anxiety, increases confidence, and allows the patient to proceed with some meaningful daily life.

It is our policy at Hillhaven Hospice to evaluate the patient's pain and response to his analgesic program during the first 24-48 hours after admission. If the patient sleeps through the night without awakening with pain in the morning, the patient is given a regular schedule of analgesic during the waking hours. If, however, the patient awakens during the night in pain, or awakens in the morning with pain, our policy is that the patient is awakened during the night on schedule to be administered a dose of analgesic. Our experience is that the patient usually falls back to sleep quickly and his state of analgesia is maintained throughout a 24-hour period.

SELECTING THE ANALGESIC:

Selection of the class of analgesic is based on the intensity of the patient's pain. In mild pain, a non-narcotic analgesic, such as aspirin or acetaminophen, can be effective in control of discomfort. As the severity of pain increases, the selection of a narcotic is appropriate. In general, the non-narcotic analgesic acts on the peripheral nervous system, whereas the narcotic analgesic acts at the central level.[5] Utilizing these principles of drug action, it has been shown in clinical studies that there is an added effect of the two analgesic classes. Therefore, the addition of aspirin or acetaminophen to codeine can produce a significant increase of analgesia.[6] Houde et al.[6] have shown that a similar additive effect occurs when aspirin is added to parenterally administered morphine.

When the non-narcotic analgesic is not effective, the choice of a narcotic analgesic usually is one of personal preference. The effective narcotic analgesics are meperidine, methadone, hydromorphone, morphine and oxycodone. The objective, of course, is to maintain comfort and minimize unpleasant side effects such as nausea, confusion, sedation, and constipation.

THE BROMPTON MIXTURE:

A most interesting and effective analgesic that is gaining increasing use in the United States is an elixir of morphine sulfate. Although most physicians have been taught that morphine is not an effective oral analgesic, Doctors Cicely Saunders and Robert Twycross have proved that oral morphine is an effective analgesic for the treatment of terminal cancer pain.[3,4] Thus, at Hillhaven Hospice, oral morphine is our drug of choice in treating cancer pain. All patients on morphine have been maintained on the oral form until the last few hours of life, or until the oral medication can no longer be ingested.

Morphine has been used in the treatment of terminal cancer pain for years in the form of the Brompton mixture. It was developed at the Brompton Chest Hospital in England and was found to be effective as an analgesic and as a dyspnea reducing agent. Over the years, there have been many modifications of the mixture with the most popular containing a combination of

morphine sulfate (or heroin), cocaine, ethyl alcohol, and flavorings. Recent studies by Twycross at St. Christopher's have produced a number of modifications: he found that morphine is as effective as heroin, with heroin being about twice as potent.[9] The addition of cocaine is not necessary for the analgesic action and may add to the side effects. Therefore, in many hospices, the *modified* Brompton mixture now includes only morphine sulfate, ethyl alcohol, and a flavoring such as grenadine syrup.

The modified Brompton mixture has many advantages over the currently available narcotics. One, it is in oral dosage form. Two, it is liquid and, therefore, easily swallowed by the debilitated patient. Three, it allows for easy increase or decrease in the amount of morphine without the patient being concerned about the change in number of tablets or change in color or shape of pills. Four, the analgesic "cocktail" can be further modified by the addition of syrups, of Compazine when nausea is a problem, or a liquid phenothiazine tranquilizer when anxiety is a significant part of the patient's symptom complex. In other words, the patient receives "one dose" of a medication that has the potential for treating several aspects of the pain syndrome. The patient's favorite alcoholic beverage can also be added and is joyfully received by some.

The mixture can be produced by any pharmacist by weighing the appropriate amount of morphine sulfate and making to volume with the addition of ethyl alcohol, water, and grenadine syrup or other flavorings. At our hospice, the concentration is 1 mg. of morphine sulfate per cc. of fluid. This allows for easy dosing of morphine by cc's. The drug is stable for several weeks in this form and is pleasant tasting.

Several studies have been done to determine the equieffective doses of oral and parenterally administered morphine. Depending on whether the studies measured peak analgesic effect, or total analgesia, the comparative dose of intramuscular to oral varies from 1:2 to 1:6. The hospice literature suggests that, on a preventive protocol, the oral dose of morphine is approximately twice that of the parenteral dose.[3,4]

Mild to moderate pain requiring a narcotic analgesic of the morphine group usually requires a dose of approximately 2.5 to 10 mg. every four hours. This dose may have to be titrated downward towards 2.5 mg. in the older patient who may tolerate the medicine less well, and increased to a point that analgesia is maintained for a period of four hours. This dose is given on a regular every-four-hour schedule; it is rarely necessary to give the dose at less than four-hour intervals. The dose is increased in increments of 5 to 10 mg. depending on our experience with a particular patient. Patients with rather severe, chronic pain, may require doses of 50-75 mg. of oral morphine or more every four hours.

At Hillhaven Hospice, with the staff experienced in the use of the modified Brompton mixture, a protocol is used to allow the nurse flexibility in adjusting the dose to maintain the patient in a pain-free state. The side effects of the modified Brompton mixture are those of morphine. The side effects of oral morphine are similar to those of parenterally administered drugs and I refer you to textbooks of pharmacology for greater detail. The most frequent side effects we encounter are mild sedation, confusion in older patients, and occasionally nausea. The most important side effect of chronically administered analgesics is constipation, which may lead to nausea and loss of appetite. Thus, a vigorous attempt at maintaining bowel function is started when narcotics are added to the symptom control program. This usually takes the form of a stool softener plus the intermittent use of a laxative or an enema.

ADDICTION AND TOLERANCE

The problem of addiction in terminally ill patients is academic and will not be discussed here. A patient in chronic pain who receives the medication for pain relief on a preventive schedule and not for its euphoric effect, is not a good candidate to become addicted. The question of tolerance is often raised when one starts to use a narcotic. Unfortunately for the patient, this has often led to inappropriate withholding of an effective drug. The studies at St. Christopher's do not support a significant development of tolerance in the terminally ill cancer patient on the Brompton mixture.[3,4] Patients have been maintained on oral morphine for upwards of one year without significant development of tolerance. The British feel that the increasing dose that may be necessary during the terminal phase of life is due not to the development of tolerance but to a progression of the underlying cancer disease. This is supported by the frequent observation that a number of patients require a decrease of dose with time. A significant development of tolerance does not permit a decrease of dose (but a changing physiology of pain production may). To date, our experience at Hillhaven supports these findings.

When an oral agent can no longer be administered because of swallowing difficulties or because the patient lapses into coma, an injectable analgesic may be required. It is our preference to change from the modified Brompton mixture to injectable morphine sulfate. The parenteral dose is approximately one-half that of the orally administered dose. Our experience with this is rather limited since the majority of our patients have been maintained on oral medications till death. I would suggest a good clinical judgment is in order in selecting the parenteral dose. A dose of up to 10-15 mg. of injectable morphine is recommended depending on the severity of pain. This can be rapidly titrated upwards or downwards depending upon the obvious response.

Nausea has not been a significant problem in our patients on oral morphine. Generally, low doses of morphine, up to approximately 20 mg. per dose, produce little nausea but this seems to be patient dependent. It is usually our policy to add 5 mg. of Compazine to the mixture when morphine doses reach 20 mg. and above, if nausea is significant. I think, again, an individual patient should be assessed with respect to the side effect of nausea

and treated symptomatically when it occurs, rather than to use an antiemetic when it is not indicated.

CONCLUSIONS:

The pain of terminal cancer has many aspects in addition to the obvious physical component that must be recognized and treated appropriately. The use of a preventive analgesic program will insure a relatively "pain-free state." Elixir of morphine is a palatable and effective analgesic that can be used without concern for significant development of tolerance.

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Emotional Disorders

The Middle-Aged Man

William B. McGrath, M.D.

Middle-aged men can be separated arbitrarily into three groups.

The blue-collar worker will have attained journeyman status fairly early in adult life. On the graph of material success his will be a horizontal line. His interests and activities may remain rather earthy: hunting and fishing and bowling, watching television and drinking beer.

A man born to affluence will tend to retain the recreations and enthusiasms of his college fraternity: football games and golf, perhaps some casual girl-chasing and the imbibing of martinis. (The incubation period for physiological addiction to alcohol appears to be about fifteen years).

The self-made man has pulled himself up from poverty or obscurity. His struggle for success will have crippled his capacity to relax and enjoy himself or simply to have fun. He is most likely to have the so-called "Type A" personality (though we object to the mutually exclusive, A or B, dichotomy).

The self-made man may then be especially vulnerable to diseases which seem to be related to stress. The relationship is not unidirectional, cause and effect. Unknown constitutional factors may generate both the person's unrelenting ambition and the physiological price he pays for it.

Assume now that such an individual has survived a coronary thrombosis or any other illness in which the prognosis hinges on his taking better care of himself.

Everyone, including his physician, will begin giving him hackneyed advice, telling him things that he has told himself a thousand times, pleading and remonstrating and scolding. "You've got to cut down your work load and your responsibilities. You should take up a hobby. You

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need to budget your time so that you'll have more regular rest and recreation and graduated exercises. Your health must have first consideration. We all care about you. We just want you to be all right."

To our angry dismay many of these patients stubbornly, almost defiantly, ignore our pleas and warnings. They keep on working and worrying, smoking and drinking, shoving against obstructions which they themselves have manufactured. They are like salmon, writhing and squirming upstream toward their icy tomb.

We react: "Well, if he wants to kill himself, that's his business and there's nothing more that we can do about it."

Not so! When we witness self-destructive behavior in patients and friends we owe it to them to find out what is going on in their minds. Offered an opportunity to share their private thoughts with us, usually at a second interview, a surprising number of persons will finally divulge sad and secret predictions: Others will benefit by their premature death; they had rather die soon than become burdensome or objects of pity. The patient with emphysema, foreseeing a prolonged and miserable doom, may persist in smoking to hasten his departure. The man who has had a stroke looks forward to nothing except deterioration and receding dignity. One wants to "quit a winner" and be remembered so.

These are lonesome and gloomy ruminations of any individual who has passed his prime. The self-made man, who never had time or encouragement to enjoy life, can anticipate even less happiness than he has heretofore permitted himself to embrace. With incomplete resignation he realizes the inevitable lessening of his personal worth, the sagging of his mental acuity and his whole physique. Taedium vitae: An older man, sage and cynical, stated his case, "I'm not tired of living; I'm tired of dying."

Before giving bromide advice to such a person we have to initiate a serious conversation. It goes something like this.

First we will not be put off by his sardonic attitudes toward himself and toward life in general. Secondly, we have no interest in such labels as, self-pity or histrionics or "copping out" or depression.

Then we point out that there is a fundamental, existential problem which he and we must try to resolve. And the answer, one way or another, will determine the kind of guidance we'll have to offer.

The issue is simply: "Do you really want to live?"

No-one except the physician will ever know how to ask that question or to elicit a considered reply. Yet every other question is secondary or derivative. And any presumptuous advice is futile and alienating.

Individuals will seldom volunteer their private thoughts. Yet every human being in the world has sometimes yearned for someone wise enough and caring enough to detect the hidden nature of his concerns and let him, just once, really express himself.



Anorexia Nervosa: A Self Imposed Disease

Stephen E. Dippe, M.D.

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Anorexia nervosa has fascinated physicians for centuries. The disease is frequently obvious to everyone but the patient. The hallmark of this disease, quite obviously, is the enormous weight loss the patient develops. The weight loss is so great that they may appear emaciated or in a state of severe starvation. The significance and seriousness of anorexia nervosa is well known to all. The big problem is attempting to get the patient to realize the seriousness of the disease. It attacks basically young females; very infrequently males. Somewhat unusual is the fact that they often appear hyperactive or "bubbly" and energetic and yet look very ill. It's thought by many that these young gals have a distorted perception of their size and shape.[1] Many of these patients suffer from a series of signs and symptoms such as constipation, hypothermia, cold sensitivity, bradycardia, and many have hypotension.[2] Interestingly,

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there appears to be a distinction between social classes as to who develops this disease. It's uncommon to find anorexia nervosa in the lower socio-economic groups. Very often these young ladies are very ambitious. Some of them have previous histories of being overweight and due to the psychological trauma of being overweight go on a starvation diet.[3] Unfortunately, they stay on this habitual starvation diet. Many individuals attempt to conceal their eating habits by vomiting purposely, hiding food, or just plain not stating the facts honestly.

They often have a very bizarre preference for food.[4] They seem to have a distorted idea as to the calorie content of food and are overly conscious of calories. They are very sensitive to even a slight elevation in weight. A few patients actually develop a state of starvation with edema.

The problem with anorexia nervosa is thought to be psychiatric in origin, but the possibility that a defect within the hypothalamus exists.[4] These young ladies do have abnormal hormonal function which can usually be demonstrated in all cases. Amenorrhea is very frequent in this disease and perhaps need be present in order to make the diagnosis in females. Associated with amenorrhea are low levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH).[5] Obviously, an appearance like starvation can occur in patients with hypopituitarism. To distinguish anorexia nervosa from patients with pituitary failure certain symptoms and signs are readily available. Both situations have amenorrhea. However, because of the lack of ACTH, cortisol levels are low in pituitary failure. In anorexia nervosa the cortisol levels are normal and in fact, frequently elevated. Thus, a person who appears malnourished with adequate thyroid and cortisol levels rules out primary pituitary failure as the cause of cachexia. Selected loss of FSH and LH occurs but is extremely rare. They usually have primary amenorrhea and do not have the clinical appearance of anorexia nervosa.

An interesting association with anorexia nervosa and cortisol metabolism is the discrepancy between the serum levels of cortisol and the urine levels of cortisol. They very often have a normal or high level of serum cortisol, and an intact circadian rhythm. However, the twenty-four hour urine steroid level is diminished. Thus, high blood cortisol levels can exist even though the 24-hour urine steroids are low. It's thought this perhaps may be associated with disrupted thyroid function.[6]

The T4 value in a person with anorexia nervosa usually is within the normal or low normal range. Their TSH values are also normal. Recent data have indicated that the T3, that is the true T3 (not the T3 uptake) is low. Whether or not these patients are truly hypothyroid remains in question. They do feel cold, have reduced body temperature, and may even have very dry skin, but they are not hypoactive and do not have many other signs of hypothyroidism. Recently a study demonstrated a low urine androsterone/etocholanolone (A/E) ratio in persons with anorexia nervosa. This low ratio is often seen in persons with hypothyroidism.[7] When T3 was administered to these patients the A/E ratio shifted upward.

Some studies have also demonstrated a high level of growth hormone.

Little is known on the exact mechanism that controls hunger. There appears to be a feeding center in the lateral portion of the hypothalamus and a satiety center in the ventromedial area of the hypothalamus. Injuries to these centers can affect anorexia and hunger. A hypothalamic tumor presenting as anorexia nervosa has been reported.[8] Regardless of what the etiology is, treatment is essential.[9] Treatment of this disease, to say the least, is difficult. If the state of starvation is severe, forced nutrition is essential. Instruction as to the quality and quantity of food may be helpful in alleviating fears. Attempting to rectify any significant psychological and psychiatric defect should be attempted. Psychiatric therapy may be of benefit. It can be truly life threatening and death will occur in many cases unless this process is interrupted.

Without doubt, medical practitioners will have an exceedingly hard time convincing the patient that they have a disease when their perception indicates that that is not the case.

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Seminars in Gastroenterology and Liver Disease

The Irritable Colon

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The irritable colon syndrome (I.C.S.) is one of the commonest gastrointestinal afflictions of mankind. It can be defined as bowel dysfunction for which no organic cause can be found. There are many synonyms for the disorder including functional bowel disease, nervous diarrhea, functional diarrhea, mucous colitis, spastic colon, irritable bowel syndrome, psychogenic diarrhea and colonic enterospasm. Many of these are misnomers, as spasm is not present in many patients, diarrhea does not always occur, and inflammation is not present.[1] Clinically, two types have been distinguished, the spastic colon, in which pain is present and often there is alternating diarrhea and constipation, and painless diarrhea.[2]

Patients with I.C.S. may represent up to 70% of patients referred to a gastroenterologist. It occurs twice as frequently in females as in males, and presents most commonly between ages 20 and 60. It should not be diagnosed if its original presentation is in individuals in their senior years.[3] In women it usually begins before age 40 but 42% of men had the first symptoms after age 50.[4]

Symptoms are present for many years before the patient seeks medical attention, with men usually waiting longer before going to a physician than women. The spastic colon variety is more common than the diarrhea form. Pain is usually present in the lower abdomen or in the left upper quadrant, but at times can be located anywhere in the abdomen, in the chest or in the back. It is often present in more than one location. It can be cramping, sharp, stabbing, burning, and either continuous or intermittent. The pain is often precipitated by eating and commences 30-60 minutes later, but occasionally while the meal is being consumed. The cramping component may be relieved by the passage of stool. Associated symptoms can include postprandial distension, heartburn, flatulence, anorexia, nausea and vomiting. Fatigue, depession, anxiety and insomnia can accompany the bowel symptoms. Diarrhea can alternate with constipation, either in the same day, or either can be present for a varying period of time to be followed by the other for an indefinite period. Mucus is present in the stool, or is a solitary rectal discharge, in 16%.[4] Twenty-two percent have been regular laxative users prior to the onset of symptoms.[2] Many have had previous surgical procedures, particularly gynecological surgery, without improvement. Some patients have the onset of symptoms following an infectious diarrhea.

The "painless" diarrhea is often associated with emotional stress. Two-thirds of patients may have cramps prior to defecation. The diarrhea characteristically is present in the morning with three or four movements not unusual before noon, and the patient is well the rest of the day, with perhaps one more evacuation occurring in the evening. The first stool of the day is often formed, but the remainder of the stools are usually loose. One-third of patients experience significant weight loss.[4]

Routine laboratory blood work is normal. The stool does not contain blood, ova or parasites, or an infectious organism. Sigmoidoscopic examination is usually normal. Spasm on advancing the instrument may reproduce the patients pain, as can air insufflation, and the instrument may not be able to be inserted to 25 centimeters. Mucus may be present. The barium enema may reveal hypermotility, a narrowed lumen, and increased haustrations with segmental spasms. The latter is particularly

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common in the spastic colon and can result in delayed evacuation of the barium. Abnormal motility may also be present in the small intestine. Absence of organic disease, however, is consistent with the diagnosis of I.C.S.

Etiology and Pathophysiology

Psychologic factors are often manifest in patients with I.C.S. In patients rigidly screened for psychiatric illness, 72% of patients with irritable colons had manifestations of hysteria or depression whereas 18% of controls exhibited similar tendencies. The psychiatric manifestations often appeared first, and the irritable colon symptoms were considered a secondary phenomenon.[5] Anger, resentment, hostility and anxiety are often associated with the spastic colon. These are associated with enhanced intestinal motor activity. Fear, dependency and dejection are associated with diarrhea, and an atonic colon on manometric studies. Women are found to worry about their families and "trifling" problems, while men are concerned more about their jobs or careers.[1] These anxieties may reflect the type of response patients will exhibit.

The diet may play a role in patients' symptoms. A low fiber diet, characteristic of Western civilization, appears to be associated with increased colonic contractions and constipation. Subsequent consumption of fruits, wheat products, nuts, milk and chocolate often exacerbate symptoms of the spastic colon. A large meal, coffee and tobacco may also enhance symptoms.

Colonic motility studies reveal enhanced motor activity, higher mean wave amplitudes and these are of greater duration in patients with spastic colon than controls. Patients with diarrhea have significantly less response in these parameters than the other two groups.[6] Within three minutes of starting a meal, there is a marked increase in sigmoid pressure in patients with diarrhea, which returns to resting levels ten minutes after completing the meal. No consistent change was found in patients with constipation. This may be related to greater propulsive activity more proximally in the colon.[7]

Three and six cycle per minute (cpm) electrical activity occurs in the colon in all individuals. Three cpm activity is more frequent in patients with I.C.S., but at basal levels there is an increased contractile activity. Following intravenous cholecystokinin or pentagastrin administration there is a significant increase in 3 cpm activity, but not 6 cpm, and the onset of contractile activity. Eating, therefore, with the liberation of intestinal hormones, may result in increased bowel motor activity, and the patients with I.C.S. may be unusually sensitive to this response.[8] Although cholecystokinin and gastrin may be involved in producing symptoms of I.C.S., the vagus nerve and serotonin appear to not be involved.[1]

Treatment: Patients with the spastic colon variety can have symptomatic response to the use of bran or bulk laxatives, such as Metamucil, and cramping and pain may improve with antispasmodics. The bulk laxatives may reduce the increased colonic pressure associated with constipation and often result in more regular bowel habits. Anticholinergics can be helpful in reducing cramping, although a 35% placebo effect has been reported.[9] Experimentally, little effect is produced on colonic motility by oral doses of anticholinergics, but clinically a response is frequent.

Antispasmodics have little role in the diarrhea form of the disease. Bulk agents are occasionally useful. Antidiarrheal agents, such as Lomotil, Imodium or codeine in limited doses are often effective.

Psychotropic agents are often helpful. A mild tranquilizer can be very beneficial if the individual is obviously anxious. Amitryptaline was found to result in improvement in a large percentage of patients in whom this drug was employed.[4]

General care includes informing the patient that he does not have a life-threatening disorder, that cancer is not present and is not a sequela of this condition, and that the life expectancy is normal. The most important aspect is to provide a sympathetic ear so that the patient realizes that he has a physician who has an understanding interest in him.

Prognosis: Patients who have obvious neurotic or psychiatric tendencies tend to have a poorer prognosis than those who do not have such obvious problems. The outlook is better if the symptoms are primarily diarrhea, or if the disease began following an infectious diarrhea. When studied long-term, the symptom pattern does not change much, but most "felt better and were more able to cope with both their symptoms and life in general. Many expressed their gratitude that someone had at last made an attempt to understand them and did not regard their symptoms as imaginery."[10]

Summary: The irritable colon syndrome is a common disorder in which abdominal pain, constipation and/or diarrhea occur for which no organic cause can be found. Employing sympathetic concern and judicious use of drugs can result in satisfying return to a more normal existence for the patient. "Peace in the colon, as well as in the world, is elusive, but worth pursuing."[1]

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Neurology

The Toxicity Of The Major Anticonvulsants

Kathern L. Plenge, M.D.

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Anticonvulsants are commonly used by physicians in all specialties, as well as general practice. Their side effects are often less well known than their indications and for this reason the toxicity of six major anticonvulsants will be reviewed. These include diphenylhydantoin (Dilantin), phenobarbital, ethosuximide (Zarontin), primidone (Mysoline), clonazepam (Clonopin) and carbamazepine (Tegretol).

Diphenylhydantoin

Dilantin is a primary drug for all types of epilepsy except absence seizures. The acute central nervous system effects attributed to oral overdosage are related to cerebellar and vestibular dysfunction and include tremor, ataxia, nystagmus and diplopia. These symptoms may persist for varying periods of time and may be minimized by lowering the dose. Acute intoxication by the intravenous route, as when treating status epilepticus, may result in hypotension, CNS depression or cardiovascular collapse. These side effects can often be prevented or minimized by decreasing the speed of intravenous administration. Intravenous dilantin should not be injected at a rate greater than 25-50 mg./min.

Subacute and chronic effects of DPH intoxication are also primarily dose related and may include ataxia, vertigo, dizziness and nystagmus associated with the behavioral abnormalities of exhilaration, euphoria, depression or drowsiness. Seizure frequency may increase. This complex of signs and symptoms has been recognized in varying contexts as "diphenylhydantoin

From: St. Joseph's Hospital & Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013.

encephalopathy."[1] This can be reversed by reduced dosage except for some unusual long-term cerebellar dysfunction following chronic overdosage.

Peripheral neuropathy has been reported in 7-29% of patients receiving long-term therapy with DPH. Clinically the signs of peripheral neuropathy are rare, but deep tendon reflexes at the knee and ankle may be absent. EMG's in the areflexic patients show slowed conduction velocities along with prolonged distal sensory latencies. Although the mechanism of this is uncertain, a possible relationship with folate depletion is under investigation.[2]

Gastrointestinal complaints including nausea, vomiting, epigastric pain and anorexia occur and can be minimized by taking the drug with meals or in divided doses.

Gingival hyperplasia occurs in about 20% and is probably the most common toxic effect of DPH in children and adolescents. The mechanism of the hyperplasia appears to involve altered collagen metabolism. It can be minimized by good oral hygiene and usually does not require withdrawal of medication. It is not dose related.

Metabolic and endocrine effects of DPH have been reported. Hirsutism is an annoying untoward effect in young females. Hyperglycemia and glycosuria have been reported due to inhibition of insulin secretion. During the past decade attention has been drawn to adverse effects of DPH on bone metabolism. Osteomalacia, hypocalcemia and elevated alkaline phosphatose have been attributed to both altered Vitamin D metabolism and inhibition of intestinal calcium absorption. Some studies indicate that treatment with Vitamin D is beneficial.

Hematological reactions include neutropenia, leukopenia, thrombocytopenia, agranulocytosis and aplastic anemia. Megaloblastic anemia is rare and responds to folic acid administration.

Hypersensitivity reactions include systemic lupus erythematous, hepatic necrosis, lymphadenopathy and morbilliform skin rashes including Stevens-Johnson syndrome.

DPH may cause orofacial dyskinesias, limb chorea and dystonia. Asterixis has been reported in patients on DPH as well as other anticonvulsants (carbamazepine, primidone and phenobarbitone).[3]

The teratogenic effects of DPH are being studied. The fetal hydantoin syndrome consists of a variable pattern of altered growth and performance and includes unusual facies, distal phalangeal hypoplasia and other defects such as cleft lip and palate.[4,5] Cardiac defects appear in a twofold to threefold increase in the fetus exposed to anticonvulsants in utero.[6] The benefits of hydantoin agents during pregnancy should be carefully weighed against their potential ill effects on the fetus.

Phenobarbital

Phenobarbital is the least toxic, the least expensive, and one of the most widely effective and widely used anticonvulsants. Seizure disorders most apt to be benefited by phenobarbital are focal seizures, grand mal seizures and febrile seizures. Drowsiness is the most frequent initial undesired effect but tolerance usually develops during chronic administration. Ataxia may occur with the drowsiness and may improve with development of tolerance or may require reduction in the dose. Nystagmus on lateral gaze is the most common motor disorder.

Paradoxical excitement[7] of the nervous system is seen in some patients. Irritability and hyperactivity can be seen in children and confusion and delirium can be seen in the elderly.

Hypersensitivity reactions of phenobarbital include scarlatiniform or morbilliform rash and rarely drug fever, agranulocytosis, aplastic anemia and exfoliative dermatitis. Hypoprothrombinemia with hemorrhage has been observed in the newborn of mothers receiving phenobarbital and Vitamin K is indicated for prophylaxis. Phenobarbital is capable of precipitating attacks of acute intermittent porphyria and is contraindicated in this disorder. Hepatitis and jaundice may occur and probably represent an allergic reaction.

Megaloblastic anemia that responds to folate and osteomalacia that responds to high doses of Vitamin D occur during chronic phenobarbital as well as chronic Dilantin therapy.

The withdrawal of phenobarbital from epileptic patients may lead to an exacerbation of seizures that sometimes proceeds to status epilepticus.

Primidone

Primidone has the same structure as that of phenobarbital except that the barbiturate ring is reduced at the 2 carbon position to form a desoxybarbiturate. Primidone (Mysoline) is most effective in the treatment of psychomotor and generalized seizures. Primidone is converted into two major metabolites: phenylethylmalonamide (PEMA) and phenobarbital.[8] PEMA also has anticonvulsant properties. Toxic reactions that occur with phenobarbital have been discussed and only those from PEMA are detailed.

Central nervous system reactions include sedation, ataxia, nausea and vomiting, personality change and radiculitis. Sedation may be dose related or the result of individual susceptibility. Personality changes show a predominance of paranoid trends but acute confusional psychoses have occurred as well. These symptoms usually remit when primidone is withdrawn. Ataxia and polyradiculitis were reported in two children, both of whom responded to treatment with corticosteroids.

Hematologic reactions include folic acid deficiency and rarely megaloblastic anemia. It has been speculated that primidone interferes with tissue metabolism of folic acid. The anemia usually responds to folate or B12, even with continued dosage of primidone. Rare cases of Vitamin K-dependent hemorrhagic disease of the newborn with maternal ingestion of primidone and thrombocytopenic purpura have been reported.

A benign maculopapular rash occasionally occurs within the first two weeks of therapy. It usually remits with withdrawal or lowering of the dose. Primidone can usually be restarted after a few weeks without a

recurrence of the rash. One fatal case of dermatitis bullosa has been reported. A syndrome similar to systemic lupus erythematous and a lymphadenopathy with malignant appearance in node biopsy have been described.

Common side effects include sedation, vertigo and dizziness, nausea and vomiting, ataxia, diplopia and nystagmus. These side effects are seen early, tend to remit with continued therapy and frequently can be minimized by starting treatment with low doses with gradual increases. It is recommended the serum primidone level be kept below 10 ug/ml.

Ethosuximide (Zarontin)

Ethosuximide (Zarontin®) is known chemically as 2-ethyl-2-Methylsuccinimide. It is considered by most physicians to be the drug of choice for control of absence (petit mal) seizures. Gastric disturbances are the most commonly described side effects and include gastric distress, nausea, vomiting and anorexia.

The next most common are central nervous system symptoms including fatigue, lethargy, headache, dizziness, hiccups and euphoria. Parkinsonian symptoms and photophobia have been described.[9] These symptoms are dose related and clear with reduction or termination of medication.

Non-dose related side effects resemble those described with other anticonvulsants. The skin and hematopoietic system appear to be the two target organs. Nonspecific skin rashes, erythema multiforme bullosa (Stevens-Johnson syndrome), systemic lupus erythematous and lupus-like symptoms have been reported.

The hematologic changes that have been noted in isolated cases include thrombocytopenia, leukopenia (12 cases) and pancytopenia (6 fatal cases).[10] Therefore, it is useful to perform periodic blood cell counts during therapy with ethosuximide and to be alert to signs and symptoms of bone marrow depression.

Behavioral changes associated with ethosuximide have been described. Negative effects such as delusions and hallucinations, anxiety and depression, and depersonalization have been observed, although positive effects have also been reported with patients exhibiting an improvement in intellectual function and psychometric performance.

Carbamazepine

Carbamazepine (Tegretol) is the only iminostilbene currently marketed for use as an antiepileptic agent. It is particularly effective in the control of focal (including psychomotor) seizures but it is also beneficial in the treatment of generalized (grand mal) convulsions. The more frequent untoward effects of carbamazepine include diplopia, blurred vision, drowsiness, dizziness, nausea, vomiting and ataxia. Some tolerance develops to the untoward effects if therapy is initiated and adjusted with care. Skin rash occurs in 3% of patients and exfoliative dermatitis and SLE have been reported.

The more serious side effects include aplastic anemia, thrombocytopenic purpura, heptocellular and cholestatic jaundice and cardiovascular collapse. Transient and spontaneously remittent leukopenia and thrombocytopenia are known to occur early in therapy.[11] Periodic complete blood counts and measurement of BUN and SGOT are recommended for patients on carbamazepine.

Clonazepam

The benzodiazepines were introduced into clinical practice in the late 1950s and are used most frequently for their anti-anxiety and muscle relaxant effects. The two that are approved as anticonvulsants in this country are diazepam (Valium) and clonazepam (Clonopin).

Clonazepam has proved effective in a wide variety of the generalized epilepsies including infantile spasms and other forms of myoclonic seizures, absence attacks, akinetic seizures and generalized convulsions. The side effects seem to be limited to the sedative action common to all benzodiazepines. Drowsiness, tiredness and hypotonia are common in the first days of treatment, sometimes with irritability and ataxia.[12] These effects disappear or are considerably reduced after one or two weeks. A very gradual dosage increase seems to prevent severe side effects of clonozepam.

Side effects occur in some patients taking anticonvulsants but the majority of patients do quite well. The clear benefit in seizure reduction warrants their use. A single anticonvulsant should be used initially and if this fails to control the seizures, a second or third drug can be added. The best use of these medications requires not only knowledge of their indications but also their side effects.

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Obstetrics and Gynecology

Drugs In Pregnancy

Part II

David Reid, M.D.

Donald J. Ziehm, M.D., Editor

Effects of drugs:

Adrenal steroid hormones, frequently used in treatment of chronic disease, are a potential problem in the fetus. Steroids have an androgenic potential and can cause genital malformations, primarily masculinzation of the female fetus. The newborn may exhibit varying degrees of clitoral enlargement and labial fusion if the drug is given between the eleventh to thirteenth week of gestation. Similar deformities can also develop from androgens administered later in pregnancy. The possibility of cortisone producing cleft palate and other head and neck deformities has been reported, primarily in animal studies. Sporadic cases have occurred in humans following administration of Prednisolone. However, it is thought that Prednisolone would be more likely to cause low birth weight, still-births and occasionally anencephaly if given in large doses. In addition to the possible toxic effects of corticosteroids, fetal adrenal function may also be suppressed. Should this occur, the fetus would encounter difficulty adapting to stress in the first week or two of life.

Hypoglycemic agents: Although insulin freely crosses the placenta, teratogenic effects have not been reported. On the other hand, oral hypoglycemics may produce congenital abnormalities and therefore should not be used during pregnancy. An infant born to a woman requiring hypoglycemic medication must also be watched carefully for the development of hypoglycemia.

Thyroid: Thyroxin readily traverses the placenta but does not seem to affect the fetus. The anti-thyroid drugs,

From: University of Alberta, Dr. Reid, Clinical Professor, Ob-Gyn.
Presented at Phoenix Ob-Gyn Society 10/20/77.

however, are of concern. The function of the fetal thyroid-pituitary axis may be altered. Propylthiouracil may produce a goiter in the fetus, but it will usually resolve following removal from the medication. The fetal thyroid can be completely ablated by I^{131}. Iodine, present in many medications taken by the mother for treatment of asthma, may produce fetal thyroid enlargement.

Alcohol: the fetal alcohol syndrome is well described. Intrauterine growth retardation, impairment of psycomotor development, microcephaly, joint changes, small eyes, small palpebral fissures — all have been documented. Recent literature suggests that the joint changes in a fetus born to a chronic or severely alcoholic mother are probably due to decreased movement resulting from the stuperous state produced in the fetus. Thus the joint anomalies may well be caused by a decrease in intrauterine fetal movement rather than any direct effect on the joints by alcohol.

Antibiotics: because of their low molecular weight most antimicrobials readily cross the placenta and may reach very high levels in the fetus. Penicillin rapidly reaches therapeutic concentrations in the fetus following administration of conventional doses to the mother. Chloramphenicol can adversely affect the newborn — the "gray infant" syndrome. Tetracycline is taken up by the matrix of the bone and can cause growth impairment or growth changes. Taken in the second or third trimester, staining of teeth and hypoplasia of development of the teeth may result. Other antibiotics, such as Novobiocin, can be associated with newborn hyperbilirubinemia. Neonatal hemolysis may occur in association with administration of Nitrofurantoin. Streptomycin in high doses can lead to nerve deafness. Anencephaly could result if Isoniazide is given in the first 40 days of gestation. Sulfamaxizol (in Septra) does not achieve the same levels in fetal serum or amniotic fluid as in the maternal serum, whereas Trimethacrine is very transportable between all compartments. Of concern is that Trimethacrine is a mild antifolate, and the fetus relies on folate for rapidly increasing cell development. Due to the antifolate activity and the fact that Trimethacrine will reach equal or higher levels in fetal than maternal tissue, Septra and Bactrim should not be used in pregnant women unless no other drug is satisfactory.

Psychopharmalological agents: most of the current anti-emetics have been implicated in fetal anomalies. Phenothiazines readily cross the placenta. Both the parent compound and its metabolite are present in maternal and fetal plasma, in amniotic fluid and in the urine of the newborn if the mother has been ingesting fairly large amounts of the medication. However, in therapeutic doses malformations usually do not occur. Phenergan, used extensively with analgesics in labor, has little effect on the fetus. Lithium has been shown to produce malformations in experimental animals. The incidence of anomalies may increase as Lithium is used more extensively in patients in the reproductive age group. Valium, Meprobamate and Librium may cause hypotonia and hypothermia in the newborn. Anticonvulsants cross the placenta very rapidly and achieve equal concentration in the fetus and mother. CNS deformities, cleft lip and palate, and defects of the developing limbs have resulted secondary to administration of Dilantin. These effects are more pronounced if barbiturates are used in conjunction with Dilantin.

Cardiovascular drugs: Digoxin crosses the placenta rapidly, reaching equal concentration in fetus and mother. Anticoagulants are given primarily for treatment of thromboembolic disorders. Heparin is a large molecule, and therefore will not cross the placenta. Thus, it is the drug of choice for anticoagulation. Coumadin derivatives freely cross the placenta and its use may result in fetal hemorrhage and death. Therapeutic levels in the mother may be disaster for the fetus. Any trauma during delivery (e.g. breech extraction, traumatic forceps delivery) may lead to a fatal intracranial hemorrhage.

Anticancer drugs: most of these compounds have repeatedly been shown to exhibit fetal toxicity in experimental animals. Numerous case reports confirm this effect in humans. Aminoptyerin, a folic acid antagonist, is one of the early drugs to have been used as an abortifacient. While not always causing abortion, cranial abnormalities and defects in the long bones frequently resulted from its use. Methotrexate, occasionally used by dermatologists, can also produce severe abnormalities.

Narcotics rapidly cross the placenta and attain concentrations in the fetus sufficient to cause varying degrees of respiratory depression in the neonate.

Environmental pollutants: Agricultural pesticides, while increasing output by controlling various crop infestations, may cause a problem in our offspring. Industrial wastes contaminate our rivers and streams. Atmospheric pollution, smoke, aerosols, sprays — how do these affect the intrauterine environment?

Factors that must be remembered in the study of pharmocologic teratogens include: species variation, formation of active or toxic compounds, distribution of the drug in mother and fetus, alteration of maternal (and fetal) enzyme systems, interaction between compounds, rate of administration of drugs and their dosage, identification of drug interactions. As more drugs become available interactions will increase.

* * *

While not intended to be an exhaustive presentation of pharmocologic dangers to the fetus, the foregoing tabulation will hopefully serve as a reminder to the physician to consider the fetus when prescribing for a pregnant woman. One of the unfortunate side effects of mass media advertising is the expectation by the "consumer" that a pill is always available to help a person feel "100%" all of the time. And the pregnant woman is no exception. It is difficult to resist the patient's plea—"I know I shouldn't take any medication during my pregnancy, Doctor, but isn't there something you can give me to relieve . . . ". The mother may feel better, but the fetus will usually resent what it has been exposed to.

Editor

Clinical Use Of Granulocyte Transfusions

Andrew Charles Eisenberg, M.D.

Editors:
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Severe leukopenia (notably granulocytopenia) is associated with marked susceptibility to bacterial infection which may be overwhelming and fatal. This occurs most often in patients with acute leukemia either as a result of their disease or its treatment but more transient episodes of granulocytopenia can also occur in other patients receiving chemotherapy. Before the introduction of platelet transfusion therapy, hemorrhage was the most common cause of death in patients with leukemia. Now infection due to granulocytopenia is the most serious threat to a successful treatment outcome of leukemia.

Almost three years ago in this column, Dr. Douglas Huestis of our institution discussed the techniques of granulocyte collection and transfusion.[1] In that article, a number of questions were raised: (1) Are granulocyte transfusions of proven benefit? (2) Which patients should be transfused? (3) Which type of infections respond best? (4) How often should transfusions be given? (5) How many cells per transfusion are necessary? (6) Which collection method is best? (7) What is the role of prophylactic transfusions? In the past three years, considerable experience has been gained at the University of Arizona.

Since January 1974, we have treated 68 patients with over 1,000 individual granulocyte transfusions for 93 episodes of granulocytopenia. All patients had a granulocyte count less than 500 on entry into the study. It was not necessary that patients have proven infection and in 15 episodes of granulocytopenia patients were given transfusions in an effort to prevent infection. All febrile patients received antibiotics with some combination of gentamicin, carbenicillin, methicillin or a cephalosporin. I shall review each of the questions raised by Dr. Huestis in the light of the Arizona experience and results of several published trials.

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CLINICAL VALUE OF GRANULOCYTE TRANSFUSIONS AND INDICATIONS FOR THEIR USE

The results of six controlled prospective trials of granulocyte transfusion therapy[2-7] were recently reviewed by Higby[8] (see Table I). There were major differences in these studies with regard to clinical criteria for entering patients, blood granulocyte count on entry, source and dose of the granulocytes transfused, mean number of transfusions, and criteria of response. In general, antibiotic therapy was similar. Within each study, transfused patients and control groups varied to a greater or lesser extent with respect to underlying disease, bone marrow status, age of patients, and type of infection. In an analysis of the University of Arizona experience each of these factors has been found to be prognostically significant.

Despite these prognostically important differences, each of the controlled prospective studies, except for Fortuny's study,[4] has shown a benefit from granulocyte transfusions in terms of clearing of infection and/or improved survival. In Fortuny's study, the control group did particularly well with 76% survival suggesting that the actual utility of the transfusions was masked by the inclusion of many patients who would have responded to conventional therapy. Also, in Fortuny's series, the number of transfused cells was very low.

SELECTION OF PATIENTS FOR TRANSFUSION

The studies of Alavi, Herzig and Vogler[5-7] demonstrate that those patients receiving transfusions without early marrow recovery show the greatest improvement in percent surviving over the control group (see Table II). However, because of the serious underlying diseases, the survival improvement was usually of short duration. In our study, the patients who obtained long-term survival (months to years) were those whose underlying disease was treatable with some prospect of marrow recovery, e.g., patients with acute leukemia undergoing initial induction therapy.

In the six controlled studies,[2-7] two different groups of patients appeared not to benefit from granulocyte transfusions. One group consists of patients who probably would have survived anyway, particularly those with evidence of early bone marrow recovery. In our University of Arizona study, there were 10 patients whose granulocytopenia was due to drug therapy alone without marrow replacement by tumor. All but one of these patients were granulocytopenic for less than 10 days and all but one recovered. (The patient who died also had severe hypogammaglobulinemia and died a month after bone marrow recovery.) This group of patients received a mean of five transfusions per episode compared to a mean of 10.5 per episode for the whole group of patients in the study and probably would have survived without granulocyte transfusions.

The other group consists of patients who died in spite of granulocyte transfusions (i.e. they were the most severely ill). Various possible reasons why these patients died include the following: the transfusions were started

TABLE I. CHARACTERISTICS OF SIX CONTROLLED STUDIES ON THE EFFACACY OF GRANULOCYTE TRANSFUSIONS (MODIFIED FROM HIGBY[8]) AND UNIVERSITY OF ARIZONA STUDY

Investigator (Institution)	WBC on entry (x 10^6 /liter)*	Qualifications for study	Dose of PMN x 10^9	Leukapheresis Method**	Mean No. of Transfusion Per Patient	Response Criteria	Benefit Shown
Graw (National Cancer Institute)	500	Septicemia	5.6 20	CFC FL	4	Survival	Yes
Higby (Roswell Park)	500	2+ days without response to antibiotics	35	FL	4	Defervescence; survival	Yes
Fortuny (University of Minnesota)	500	48 hours after demonstration of infection	6.2	CFC	4	"Clearing of infection"; defervescence; survival	No
Vogler (Emory)	300	72 hours after demonstration of infection	29	CFC	7	"Clearing of infection"; survival	Yes
Alavi (University of Pennsylvania)	250	36 hours after antibiotics had been started	50	FL	7	Survival	Yes
Herzig (NCI)	1000	Positive blood cultures	4/m^2 BSA 17/m^2 BSA	CFC FL	9	"Clearing of infection"; survival	Yes
University of Arizona	500	Availability of transfusion	14	IFC	10.5	"Clearing of infection"; survival	(No control group)

* WBC x 10^6 /liter = WBC/mm^3

** CFC = continuous flow centrifugation leukapheresis; FL = filtration leukapheresis; IFC = intermittent flow centrifugation.

too late or administered in insufficient quantity; the infection may have been viral; or, there may have been some unrelated cause of death such as hemorrhage or renal failure. It has been observed, both by Higby[8] and here at the University of Arizona, that patients with fungal infection may be less responsive to granulocyte transfusions than those with bacterial infection. In the Arizona study, all six patients with a diagnosis of systemic candidiasis eventually died of infection. Additionally, postmortem studies identified three other cases where candida infection contributed to death. In all, 9 of 24 deaths (37.5%) in our series were associated with candida infection. Despite this lack of clinical efficacy in candida infection, Glasser, *et al.*, have demonstrated that granulocytes prepared for transfusion have fungicidal activity *in vitro*.[10]

TABLE II
EFFECT OF EARLY MARROW RECOVERY ON SURVIVAL SUMMARY OF CONTROLLED STUDIES OF GRANULOCYTE TRANSFUSIONS*

Patient Group	PERCENT OF PATIENTS SURVIVING (NO. SURVIVING/TOTAL NO.)** With Early Marrow Recovery	Without Marrow Recovery
Control (no WBC)	100% (15/17)	9% (2/23)
WBC Transfusion	100% (16/16)	78% (21/27)

* Combined Results: Vogler, Alavi, Herzig.[5,7] Antibiotic use comparable in all studies.

** Volger, Alavi, survival 21 days; Herzig, mean survival 3 mos.

FREQUENCY OF TRANSFUSIONS AND DOSE; METHODS OF GRANULOCYTE COLLECTION

The questions of how often transfusions should be given and how many cells per transfusion are necessary cannot be adequately answered by analysis of the controlled trials. Fortuny's study[4] may be interpreted as suggesting that doses of granulocytes in the general range of 4 to 7 x 10^9 cells are essentially ineffective. Despite the trial by Graw[2] in which levels in this range were used, Higby[8] was impressed enough by the Fortuny study to recommend that transfusions consist of a minimum of 1.5 x 10^{10} granulocytes from continuous flow centrifugation (CFC) leukapheresis and probably larger doses from filtration leukapheresis (FL). Because 1.5 x 10^{10} granulocytes represents only a fraction (1/10 - 1/100) of the potential output per day of a normal bone marrow stimulated by sepsis, granulocyte transfusions have generally been given on a daily basis (except weekends). We plan to investigate whether such transfusions given twice per day may prove more efficacious.

There are four basic methods of collecting granulocytes (see table III). The six controlled studies suggest that FL cells can be used as effectively as CFC cells in the support of neutropenic patients, despite the fact that FL leukocytes are probably less effective cell for cell. We have been particularly impressed by the advantage of intermittent flow centrifugation (IFC) leukapheresis, when used so as to provide platelets along with the granulocytes. Using daily transfusions at the University of Arizona, it has been possible to raise platelet counts to over 100,000/mm^3 and markedly diminish the danger of hemorrhage in patients who are thrombocytopenic as well as granulocytopenic. Platelets are not recovered for transfusion with CFC or FL.

ROLE OF PROPHYLACTIC TRANSFUSIONS

Two controlled prospective trials of the use of prophylactic transfusions in humans have now been published. Ford and Cullen[12] evaluated the use of granulocyte transfusion obtained by IFC given every other day in patients with acute non-lymphoid leukemia and granulocyte count less than 500 x 10^6/liter (500 cells/mm^3). In the small number of patients studied, there was no apparent effect on the clinical course of those receiving transfusions versus those in the control group. In a similar group of patients, Mannoni *et al.*[13] gave prophylactic transfusions from the 4th day of chemotherapy to the 12th day of marrow aplasia. They showed a significant decrease in documented infections in the transfused group and there were no deaths in the transfused group.

At the University of Arizona, there were 15 instances in which granulocyte transfusions were given to afebrile, clinically not infected, granulocytopenic patients. In only two patients did this seem effective — one who was granulocytopenic for six days and remained afebrile, and one who was granulocytopenic for 26 days (receiving 23 transfusions) and was afebrile until the day before he died of unresponsive multiple myeloma. The remaining 13 patients all became febrile within five days (cultures positive in five) despite prophylactic granulocyte transfusions. In addition, there were 13 instances in which patients receiving granulocyte transfusions became afebrile and antibiotic use was discontinued. Despite continued transfusions, the patients again became febrile (cultures positive in six). The recurrent fever was noted within 48 hours in all but three of these latter patients.

OTHER CONSIDERATIONS

Two important issues not raised by Huestis[1] but mentioned in an editorial by Boggs[14] are the *availability* and *cost* of granulocyte transfusions. At the present time the University of Arizona is able to provide transfusions for only four patients per day and at times this has not been adequate. The only other source of granulocyte transfusions in Arizona is Arizona Blood Services of Phoenix, which also uses the IFC technique developed by Huestis. They are able to support 4-6 patients at any given time. Aside from uses in research, the cost of granulocytes is in the range of $250 per transfusion. Therefore, it is crucial that their use be limited to those patients who can maximally benefit from this form of therapy (see Table IV).

CONCLUSIONS

Granulocyte transfusions have been conclusively shown to be of benefit in improving survival by controlled clinical trials. Patients with early bone marrow recovery probably do as well with conventional therapy alone. Although patients who do not have bone marrow recovery show the most significant benefit, their survival is relatively short, so that in the future because of limited availability and considerable cost, these transfusions may have to be limited to those patients whose underlying disease can be put into remission. Patients with invasive

TABLE III. COMPARISON OF LEUKAPHERESIS METHODS (MODIFIED FROM HUESTIS[1])

METHOD	ADVANTAGES	DISADVANTAGES
Continuous Flow Centrifugation (CFC)	Minimal trauma to cells Small extracorporeal blood aliquot Versatility: can be used for PMNs, platelets, lymphocytes, plasmapheresis	Highest initial cost Low separation efficiency More difficult operation
Intermittent Flow Centrifugation (IFC)	High efficiency for PMNs Provides PMNs plus platelets Ease of operation Disposable pathway Minimal trauma to cells Versatility: can be used for PMNs, platelets, lymphocytes, plasmapheresis, RBC washing	Moderate initial cost Large extracorporeal blood aliquot Small volume of blood per hour
Filtration Leukapheresis (FL)	Highest efficiency for PMNs Few lymphocytes Low initial cost Disposable pathway Ease of operation	Collects only PMNs and some monocytes Functional impairment of PMNs High heparin dose to donor More donor reactions More recipient reactions Variable efficiency of batches of nylon filter
Gravity Leukapheresis (GL)	Low cost No equipment needed Minimal trauma to cells Good yield of PMNs and platelets	Multiple separate centrifugations Hazard to donors from separated RBCs

TABLE IV. CRITERIA FOR SELECTING PATIENTS TO RECEIVE GRANULOCYTE TRANSFUSIONS

1. Granulocyte count less than 500.
2. Prolonged granulocytopenia anticipated (greater than 10 days)
3. Underlying disease capable of going into remission.
4. Fever (suspected infection).
5. Availability of donors and "machine time".

andidiasis do not respond well to granulocyte transfu-ions, but it is difficult to diagnose fungal infection premortem. The initial impression is that prophylactic ransfusions are not of benefit in preventing infection. The question of optimal number of cells per transfusion, frequency of transfusions, and method of collection of the cells still needs further investigation.

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Radiology

Case Of The Month # 29

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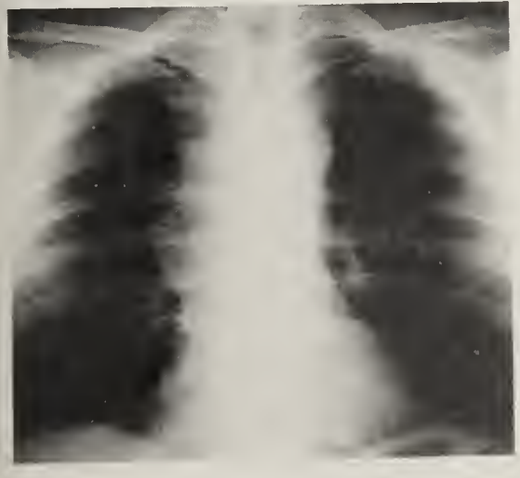


Figure I: Anterior view of 99mTc sulfur coloid liver scan.



Figure II: Right lateral view of same scan.

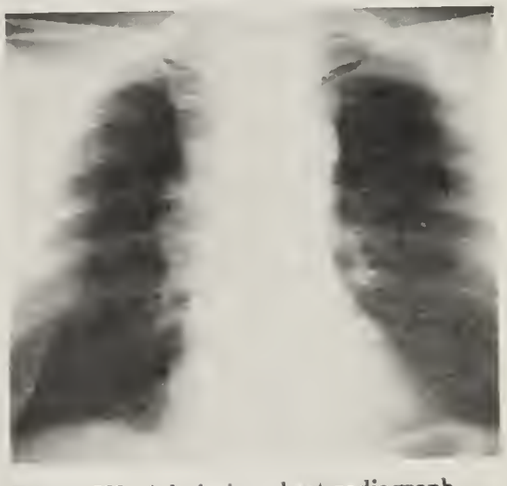


Figure III: Admission chest radiograph.

A ^{99}mTc sulfur colloid liver scan was obtained as part of the initial work-up of a 48 year old male. Note the well localized area of relatively increased tracer activity (or "hot spot") in the liver (see Figures I and II). The patient's admission chest radiograph (Figure III) demonstrates an abnormally widened superior mediastinum.

Based on these two studies, what is the most likely explanation of the liver scan abnormality?

From: U of A Health Sciences Center, Tucson, AZ 85724.

"Hot Spot" Detected on 99mTc Sulfur Colloid Liver Scan Due to Superior Vena Caval Obstruction

Space-occupying lesions of the liver, if of sufficient size, will present on radionuclide scintigrams as areas of relatively diminished radiopharmaceutical activity. These "cold spots" are the most frequently encountered focal abnormalities seen on ^{99}mTc sulfur colloid liver scans and indicate that the normal cellular uptake of the tracer activity has been displaced by a mass (e.g., neoplasm, abscess, hematoma, parasitic infestation, regenerating cirrhotic nodules, etc.). The hepatic macrophages (Kupffer cells) contain the tracer activity demonstrated on radiocolloid liver scans. These cells line the normal liver sinusoids and phagocytize the foreign particles of sulfur colloid, accumulating the tagged ^{99}mTc tracer activity.

Regions of increased tracer activity, or so-called "hot spots" in the liver, are rarely encountered. A solitary region of increased radiocolloid uptake is non-specific and has been reported in liver abscess, arteriovenous malformation, primary or metastatic hemangiosarcoma, Budd-Chiari syndrome, hydatidiform mole, and hepatoma. However, a discrete "hot spot" localized antero-medial to the porta hepatis, as in this case, strongly suggests superior vena caval obstruction. Figure III shows a widened mediastinum which is compatible with this condition.

Both isotope and radiopaque venography have shown that such a focal area of concentrated tracer activity, characteristically located superior to the gallbladder fossa on the anterior view, is the result of collateral circulation. In a patient with superior vena caval obstruction, a radiopharmaceutical bolus injected into an antecubital vein, flows through numerous collateral venous channels in the anterior and posterior soft tissues of the chest and abdomen, bypassing the obstructed superior vena cava. Approximately 25% of these collaterals anastomose anteriorly with an umbilical vein remnant which joins a branch of the left portal vein (the *portal venous ramus retroflexus*) which perfuses the central portion of the anterior aspect of the liver. Thus approximately one fourth of the injected radiocolloid bolus is delivered to a very small focal area in the quadrate lobe of the liver. It is important to recall that, although the umbilical vein and ductus venosus collapse shortly after birth, the umbilical vein retains its patency throughout adult life and can facilitate a large volume of collateral venous drainage, if necessary. The Kupffer cells in the liver sinusoids there phagocytize the majority of this tracer activity on its first pass through the liver, resulting in this well localized, characteristic "hot spot." The remaining portion of the bolus is delivered to the liver in the normal fashion (i.e., 80% by the portal vein and 20% by the hepatic artery) and results in the diffuse, more homogenous tracer activity seen throughout the remainder of the liver.

Approximately 80% of the cases of superior vena caval obstruction are due to malignant tumors involving the mediastinum. The actual diagnosis and etiology of this patient's superior vena caval obstruction was proven at autopsy to be a small focus of oat-cell carcinoma located in the right upper lobe, which at the time of this examination had metatasized to the right hilum and superior mediastinal nodes. This small pulmonary parenchymal focus of neoplasm could not be visualized on the plain chest radiograph, which is not at all unusual for this extremely malignant cell type.

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Medicine In Mexico

VII. Rural Health Care Delivery

Charles C. Dashbach
William B. McLeod

Mexico's health care structure is directed primarily towards the urban population, generally neglecting almost 20 million rural inhabitants. In 1971, a government survey reported 223 doctors per 100,000 urbanites and 3 doctors per 100,000 persons in rural areas. Rapid urbanization, a torturous geography and a lack of information concerning rural health problems all contribute to this maldistribution of health care delivery.

Forty-two percent of Mexico's total population inhabit communities of less than 2,500 persons. In these communities public services are virtually nonexistant with 15.6% of the rural population covered by one of the socialized health services (SSA, IMSS, ISSSTE, etc.), leaving 84.4% to traditional folk healers and sporadic visits by mobile brigades. The health problems of this population are considerable. Malnutrition, infectious and parasitic diseases, and complications of pregnancy are their most frequent cases of mortality.

Nationwide studies of socio-economic standards always emphasize the problems of the rural areas. Their population increase is much higher than the national average (3.2%) and their average income per month per family is less than 675 pesos (about 32 dollars). The 1970 Census reported that 40% of Mexico's families (16 million persons) lived in one room houses and a mid-1976 study revealed over 56% of all homes to be without adequate sewage facilities.

While the traditional health providers for this population will be discussed in a later article, it should be noted that this population is not totally without health care. Traditional folk healers [*curanderos*], mid-wives [*parteras*], and pharmacists play an important role in rural Mexico. Although their impact is difficult to measure, they must be considered as a viable and integral part of the rural health spectrum.

The Rural Administration branch of the Ministry of Public Health and Welfare (SSA) has targeted several areas for their current efforts. Two of these, the *Zona Huicot* and the *Altos de Chiapas*, represent the variety and flexibility needed to meet the health needs of different areas.

The program in the high mountains of the State of Chiapas was started in 1974 and is intended to serve a population of 378,614 over an area of 5100 km² of rugged mountainous terrain. A majority of this population are indigenous (Tzotziles, Tzeltales, Tejolabales, Choles and Lacandones) and the program has anticipated many of the cultural barriers to be faced. For example, the initial stages of the campaign involving the dropping of thousands of portable radios, followed by health education broadcasts in native dialects. A united effort involving six ministries, public utilities and UNICEF is attempting not only primary health care delivery, but a general stimulation of the physical, economic and socio-political progress of the area's 21 municipalities and their out-lying villages.

The *Area Huicot*, named for its three Indian tribes (the Huichol, the Cora and the Tepehuan), is inhabited by only 65,900 people (1973), but they are scattered over an almost inaccessible 27,725 km² region through the States of Durango, Jalisco, Nayarit and Zacatecas. Considerable controversy has arisen by government efforts to improve living conditions for this isolated population in the Sierra Madre Occidental. These tribes and their customs, particularly the Huichol, have resisted the encroachment of modern man for centuries and it was not until the 1960's that outsiders were allowed to enter the area. From 1971 to 1973, 26 *Centros de Salud* (each staffed by 2 doctors and 4 nursing auxiliaries fulfilling their government social service obligation) and 7 *Casas de Salud* (with one health educator and local volunteers) were constructed. As in the Chiapas program, a coordinated effort is being used which attempts to recognize particular customs and traditions.

The Mexican rural health effort is under the jurisdiction of a special branch of the Public Health Secretariat, the Director General of Public Health Coordination and Services. Table I shows the facilities and manpower statistics for this program in 1973. While the manpower/target population/facilities ratio are far from adequate, it must be remembered that this program is not budgeted nor designed to provide comprehensive health care, but rather functions primarily in a preventive and extigent role.

A modular-pyramid approach to health care resources central coordinating hospitals, rural *Centros de Salud*, and small *Casas de Salud* to maximize the effort. A theoretical structure for a rural population of 150 to 200,000 persons would have one urban coordinating hospital, 5 suburban health centers, 15 rural *Centros de Salud* and 90 rural *Casas de Salud*. It is hoped that such a system can work hand-in-hand with other government programs to raise the living standards of the rural poor in Mexico and serve as a model for other developing countries.

TABLE I
Health Care Facilities and Health Manpower of the Mexican Directorate General of Coordinating Rural Public Health, 1978[1]

Theoretical Rural Target Population: 26,724,492 (49% of total population)

Facilities:	Number of Units	Number of Beds
Coordinating hospitals	52	4763
Urban Centros de Salud	109	412
Suburban Centros de Salud (without hospital)	100	271
Suburban Centros de Salud (with hospital)	397	5185
Rural Centros de Salud	1485	4114
Guarderias y Albergues	27	56
Mobile units	22	-
Others	6	105
TOTAL	2198	13950

Personnel:	
Doctors	2470
Nurses	6846
Social Service Doctors	2473
Administrative	3149
Paramedics	2783
Intendencia (Administration—Mayor or Gov. officials)	3275
Others	608
TOTAL	21604

[1]from Direccion General de Servicios Coordinados de Salud Publica en Estados y Territorios, informe 1973.

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Accurate Measuring Of Daily Insulin Doses:

Aids For The Blind Diabetic

Michael S. Kappy, M.D.

Blindness occurring as a complication of diabetes mellitus is the leading cause of blindness in the United States.[1] It affects 7 percent of all diabetics,[2] or an estimated 250,000 people.

Recently, one of our new patients, a 28 year old blind female diabetic expessed the desire to be able to draw up her own daily insulin dose. She stated that her usual daily dose was 25 units of NPH insulin, but that she occasionally took 20 units when she exercised more than usual. With this regimen, she had been able to avoid both hypoglycemic episodes and excessive glycosuria. Her husband or her mother would prepare her dose for her each day. On occasion, she would exhibit ketonuria when ill, and would require small additional doses of Regular insulin during the day.

A search for a device which would give her the independence she wanted uncovered several of them which have been developed to enable blind and partially-sighted diabetics to accurately administer their own insulin, either singly or as a mixture of two insulins.

The Andros, Inc. (2332 Fourth St., Berkeley, CA 94710) IDM® is a thin plastic strip which may be cut off by the physician to the length which corresponds to the desired dose, e.g. 40 units (Fig. 1a). The patient fits the projection on the syringe into the groove in the plastic, and by withdrawing the plunger slightly beyond the cut end and then pushing it back to the cut end (Fig.1b) any accidental air in the syringe is injected back into the bottle, and the correct insulin dose remains in the syringe.

The Andros IDM® is available only for use with U-100 insulins and Beckton Dickinson (B-D) #8409 Plastipak® syringes. Some degree of flexibility of doses for single insulins or mixtures of insulins in the same syringe could be provided by giving the patient several strips, each cut to a different length, but the strips would have to be marked in some way (e.g. notches cut into them) for the patient to identify them, especially if their lengths were not greatly different. The strips cost approximately $2.50 apiece, and should be replaced every three months according to manufacturers instructions. Thus, the cost to a patient who needs two or three of these would be approximately $25.00 yearly (as of 10/77).

The Insulgage® system by Meditec, Inc. (9485 E. Orchard Dr., Englewood, Colorado 80110) is a series of marked plastic gages each cut to a length corresponding to an insulin dose. The patient fits the plunger of the syringe into the slot in the gage (Fig.2a) and withdraws insulin to a point slightly *beyond* where the top of the gage fits against the syringe (Fig. 2b), and then pushes back until a snug fit is achieved, thus, expelling any air bubbles and insuring the correct insulin dose.

The Insulgages® provide for flexibility of doses for single or mixed insulin regimens, and are well-marked for identification by fingertip (unit numbers are also available in Braille). Sets of Insulgages® are available for U-40, U-80, or U-100 insulins, and the B-D #8400 series Plastipak® syringes, or the Jelco #1022 syringe (U-100 only). There are about 45 different gages available, from 2 to 85 units. Sets of 1, 2, 3, 5, 8, and 12 gages are available, and are reportedly "permanent." A set of three different gages (raised numbers) is $10.00 (as of 10/77).

The descriptions provided here are by necessity incomplete. Detailed descriptions of these or other devices,[2,3] as well as specific directions for their use should be obtained from the manufacturer listed for each device, or from the following sources: 1) Arizona Diabetes Association, 555 W. Catalina Dr., Phoenix, Az. 85013, 2) Minneapolis Society for the Blind, 1936 Lindale Avenue South, Minneapolis, Minnesota 55403, 3) American Foundation for the Blind, 15 W. 16th Street, New York, New York 10011, and 4) Hypoguard Ltd., 49 Grimston Lane, Trimley, Ipswich Suffolk England IP10 OSA.

Several methods are available to the patient to estimate the amount of insulin left in a bottle, e.g. by shaking the bottle or by keeping track of doses used with marbles in a cup, etc. Our patient, taking 25 units of NPH insulin daily, could avoid accidentally drawing up excessive air and thus decreasing the actual insulin drawn, by starting a new bottle on the first day of each month.





Figures 1a and 1b — The Andros IDM® Device.

Patients with chronic illness should be encouraged to retain as much independence as possible for reasons of self-esteem and for times when they may be temporarily without help. Devices such as those described above are means whereby the blind diabetic may do just that.

ACKNOWLEDGEMENTS

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Figures 2a and 2b — The Meditec Insulage® Device.

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U of A Grand Rounds

Hirsutism

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Editors:
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Hirsutism may be the most common endocrine abnormality of women. Approximately one-third of menstruating females have upper lip hair, about ten per cent have chin hair or sideburns, and after the age of 60 about three-quarters of the women have excessive facial hair. Androgen excess supports this abnormal hair growth as well as that seen on the chest and abdomen. The medical evaluation of hirsutism has often been a source of confusion to the primary care physician, and the results of treatment unrewarding.

There are several common misconceptions about hirsutism. It is not true that it cannot be successfully treated unless a tumor or adrenal hyperplasia is found. Nearly all forms of androgen excess can be reduced with medical therapy. It is not true that hirsutism is *not* a result of androgen production. Finally, it is not true that hirsutism is a genetic disorder that does not lend itself to effective treatment. As it turns out, one of the most common causes of hirsutism, polycystic ovarian disease, usually is familial, however, treatment successes are the rule.

Testosterone (T), androstenedione (A), dehydroepiandrosterone (DHEA) and its sulfate (DHEA-S) are the major androgens produced and secreted into the circulation of the normal female. The ovary is the primary source of direct T secretion and normally contributes about 50 per cent of A production. In normal females approximately 50 per cent of the daily T production rate is derived from the peripheral conversion of A to T. In hirsute women a greater proportion of plasma T may come from direct oversecretion of T. DHEA and DHEA-S are primarily adrenal products. Hirsutism in women can be attritubtable to (1) tumors of the ovary or adrenal, (2) the adrenal cortex, (3) the ovary, or (4) idiopathic causes.

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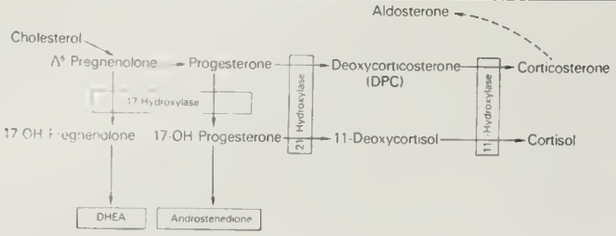


FIGURE 1
Common forms of congenital adrenal hyperplasia (CAH).

Virilizing tumors typically secrete massive amounts of androgen and can often be suspected by the rapid appearance of clinical virilization in addition to severe hirsutism. Plasma A concentrations greater than 1,000 ng/dl or plasma T concentrations greater than 300 ng/dl (both in the male range) nearly always indicate an adrenal or ovarian tumor. These are uncommon causes of hirsutism and will not be further discussed in this brief review.

The adrenal cortex can be the source of excessive androgen secretion other than in tumor states. Congenital adenal hyperplasia (CAH), either partial or complete, can present as hirsutism at the time of puberty or as an "acquired" form later in life. The terminology CAH encompasses partial or complete deficiency of one of the hydroxylating enzymes in the steroid biosynthetic pathway of the adrenal cortex (see Figure 1). Deficiency of 17-hydroxylase, an uncommon form of CAH, impairs production of all of the sex steroids and does not cause hyperandrogenism. Both 11-hydroxylase and 21-hydroxylase deficiencies cause excessive accumulation of the glucocorticoid precursors and excessive stimulation of the androgen biosynthetic pathway. The 21-hydroxylase form is the more common and can best be detected by the demonstration of elevated plasma 17-hydroxyprogesterone (17OHP) and plasma DHEA or DHEA-S (since little of these come from the ovary). The 11-hydroxylase form is distinguished by the presence of hypertension and elevation of the plasma 11-deoxycortisol, in addition to increases in plasma 17OHP, DHEA and DHEA-S. As a cause of hirsutism, CAH is far less common than the ovarian disorders, and tends to present in younger females. Treatment of all forms of CAH is aimed at stopping the excessive stimulation of the adrenal cortex by ACTH. This can be accomplished by administration of physiologic replacement doses of oral glucocorticoids (i.e., prednisone 7.5 mg/day).[1,3]

Polycystic ovarian disease (PCOD) is a common cause of hirsutism. In the last few years, our understanding of the pathophysiology in this disease has greatly increased, although its cause is still obscure. A severe form of this disorder was originally described by Stein and Levinthal. Their original description included obesity, anovulation, enlarged ovaries, and hirsutism. PCOD is actually a spectrum of ovarian disorders ranging from monocystic ovaries,[4] to hyperthecosis (replacement of the ovary by androgen secreting theca cells). Most patients with PCOD do not have palpably enlarged ovaries. They may not be obese, and may ovulate occasionally. Presenting complaints often consist solely of menstrual irregularity and mild hirsutism. The disease tends to be familial. Involved families may also have subnormal male fertility. In these less obvious cases of PCOD, the diagnosis usually is made by examining the plasma concentrations of gonadotropins and androgens.

The typical patient with PCOD has an elevated plasma leutinizing hormone (LH), a normal or low plasma follicle stimulating hormone (FSH), an elevated plasma T and A, and an elevated plasma estrone (E_1).[5] The overlap with normal female values is considerable on a single serum sample. Repeated plasma hormone examinations are sometimes necessary.

These hormonal aberrations are often quite modest on casual plasma examination, but their basis is now better understood and can be summarized as follows.[6] The high LH concentrations appear to be maintained by the presence of exaggerated pulsatile LH release, both in frequency and amplitude. Normal pulsatile FSH secretion does not occur in these patients. As evidence that the inappropriate LH levels are not due to a defect in negative feedback, it can be shown that acute intravenous estradiol infusion results in an appropriate LH fall. There is a heightened pituitary LH response to infused leutinizing hormone-releasing factor (LRF) that correlates well with the elevated basal E_1 levels. This exaggerated positive feedback appears to be similar to that seen in the normal preovulatory estradiol surge that triggers LH release and ovulation. In contrast, elevated estrogens have a preferential inhibitory action on the FSH response to LRF in addition to the inherent insensitivity of FSH release to LRF. Therefore, in PCOD the abnormalities of hypothalmic-pituitary release regulation of gonadotropin secretion are not an inherent defect, but represent a functional derangement consequent to inappropriate estrogen feedback, which leads to a cycle of chronic anovulation and inappropriate stimulation of the ovary by LH.

Whether or not the cystic nature of the ovaries is a primary disorder or a secondary event due to chronic LH overstimulation is not known. The net result is ovarian hypersecretion of T and A. This is reflected in an elevation of the plasma levels of both hormones. T is a much more potent androgen than A and most likely accounts for the majority of the hirsutism, oily skin and acne. A is more important as a precursor for extraglandular E_1 production and significantly accounts for the excess of circulating E_1 concentrations.[7] The peripheral tissue conversion of A to E_1 in PCOD may increase daily E_1 production fivefold. This increase mainly reflects an increase in the amount of substrate (A) available. But obesity itself has been shown to increase the rate of conversion, adding support to the clinical observation that obese patients with PCOD seem to have a more severe form of the disease. These hormonal aberrations are diagrammed in Figure 2.

An additional abnormality has been defined in PCOD. Androgens normally circulate in a protein-bound and a free form. It is the free fraction that is metabolically

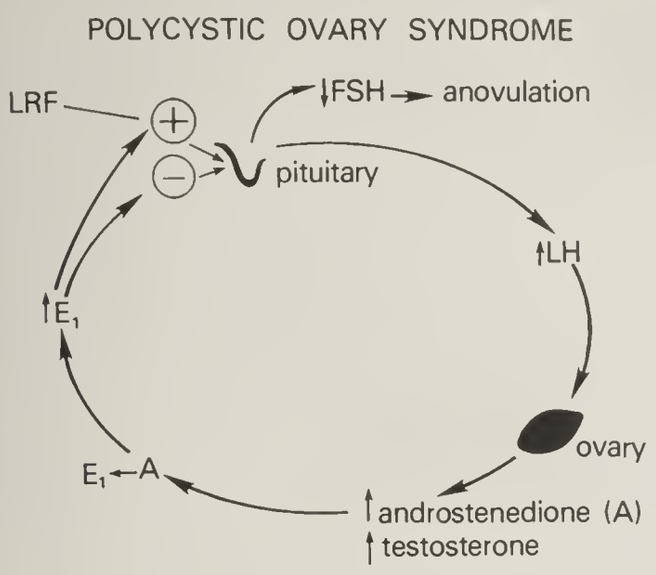


FIGURE 2
Cyclic hormonal aberrations in polycystic ovarian disease (PCOD).

active. Women with PCOD usually have a depressed level of serum sex-steroid binding globulin and consequently have an increase in the free fraction of their circulating testosterone.[8] The depressed level of sex-steroid binding protein is a result of the increased circulating androgens in PCOD.

Medical interruption of the abnormal hormone cycle in PCOD is possible in most cases. Using a combination estrogen-progestin contraceptive pill (containing norethindrone 2 mg. and mestranol 0.1 mg., Ortho-Novum® 2 mg. or Norinyl® 2 mg., or norgestrel 0.5 mg. and ethinyl estradiol 0.05 mg., Ovral® , it has been shown that plasma concentrations of A, T, LH, FSH and sex-steroid binding globulin all return to the normal range.[9] In clinical use a combination-type contraceptive pill containing less estrogen than those mentioned above can be substituted after the first two or three months of therapy. After 3 to 12 months of continuous treatment, most patients will have decreased skin oil, acne and hirsutism, in addition to regular withdrawal menses. Patients should be counseled about the expected slowness of response. The traditional ovarian wedge-resection gives only a temporary decrease in circulating androgens and seems to be an unnecessary risk (general anesthesia and intra-abdominal surgery) for a cosmetic endpoint. After a medical remission is induced optional electrolysis can remove any remaining coarse, dark hairs that have grown in an excessive androgen milieu.

When a source of androgen hypersecretion is not obvious from physical or laboratory examination the resultant hirsutism is said to be idiopathic. The classic approach to this problem has been to perform stimulation and suppression tests of the adrenal cortex and ovaries to identify the abnormal gland and select appropriate therapy. A careful analysis of the predictive value of such maneuvers finds them terribly misleading. Ettinger and his co-workers[10] examined the response of plasma T to the administration of adrenocorticotropin (ACTH), human chorionic gonadotropin (HCG) and dexamethasone. Patients were then treated for three months with either dexamethasone or estradiol-progesterone, regardless of their initial stimulation-suppression test results. Results of the short-term testing did not correlate with those of long-term treatment. Previously elevated plasma T levels were suppressed in 80 per cent of the patients treated with dexamethasone and in all the patients treated with estradiol-progesterone. Clinical improvement and normalization of plasma T levels correlated well.

One explanation for the nonspecificity of the stimulation-suppression testing is the concept of hormonal "crosstalk." That is, the notion that tropic hormones and their target glands are not completely specific. For instance, it has been shown that the cortisol secretion rate may decrease 30 per cent in normal women receiving Norinyl® 2 mg.[11] More convincing evidence of "crosstalk" is seen with the suppression of ovarian androgen production by dexamethasone.[12] This was demonstrated during the course of adrenal and ovarian vein catheterization of 13 females with idiopathic hirsutism. Nine of the 13 were found to have an exclusive ovarian source for their excess androgen secretion and 3 of these 9 suppressed this abnormal production with dexamethasone.

The concept of idiopathic hirsutism ought to be abandoned. There is excellent evidence that idiopathic hirsutism is an ovarian abnormality.[13] Forty-four consecutive women with unexplained hirsutism were investigated, 41 of whom had normal-sized ovaries. Dexamethasone suppression showed at least 50 per cent reduction in plasma T and A concentrations in 20 of the subjects. By examining ovarian and adrenal vein effluents for T and A, it was shown that 42 of the 44 hirsute women, including those whose androgens were suppressed after dexamethasone, had ovaries that were the predominant source of androgen production.

The approach to the female patient with hirsutism is now simplified. Severe and rapidly progressive hirsutism/virilism combined with plasma A and/or T concentrations in the male range strongly suggest an adrenal or ovarian tumor. Hirsutism, with or without hypertension, in a young female makes CAH a reasonable consideration, and can be confirmed by elevations in the plasma concentrations of DHEA, or DHEA-S and 17OHP. If the physical examination or family history suggest PCOD, measurement of plasma LH and T will usually confirm the diagnosis. Idiopathic hirsutism is usually an ovarian abnormality and belongs as a variant of the PCOD group.

Androgen secreting tumors should be sought by arteriography or surgical exploration, and removed when possible. CAH is effectively treated with physiologic doses of glucocorticoids. Most hirsute patients have PCOD, or a variant thereof, and should be given suppression with oral contraceptives if there are no contraindications.

Continuing evidence that users of oral contraceptives have a significantly increased risk of death from diseases

Continued on Page 196

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A Unified Voice
A Unified Action
A Unified Profession

The AMA, State, and County Medical Associations — the Federation of Medicine — can be a formidable force in shaping the destiny of American medicine.

It scarcely needs saying that the medical profession — every physician in this country — is facing tremendous challenges. The need for a unified voice and unified action by the medical profession is essential now. Federal and state intervention into the practice of medicine, control through legislative fiat, investigative harrassment, and government regulation requires an organized response emanating from a unified profession. There is an urgent need for a unified effort by the medical profession to maintain the highest possible standards of medical practice, education and research.

The federation of medicine is a unifying force that democratically blends the viewpoints of all physicians into concerted and effective action. The strength of organized medicine derives entirely from the participation and support of individual physicians. The AMA represents physicians on the national level. The State Medical Association represents the physician in statewide health care oriented problems in legislation. The County Medical Society functions within the local environment. All work together to represent the individual physician.

According to Webster, "United" means "combined, joined, or made one." Being part of organized medicine implies involvement at all levels — County, State,' and National just as citizenship implies voting and being taxed at all three levels. Wouldn't it be great to pay taxes at one level only? Unfortunately, life isn't that simple! We must pay the toll at all levels or exercise our *freedom of choice* and go to some other country. Unified membership in organized medicine is based upon sound logic: it promotes unity, it discourages splintering of resources, and demonstrates authority to the external body politic. No one can deny that we need to address the body politic in a unified manner at all levels now more than ever before.

Many of the critics of voluntary (because it is truly voluntary — you don't have to belong to any organization for licensure, hospital privileges, etc.) unified membership expound loud and long about unified membership causing a drop in state society membership. The truth is that New York, for instance, *increased* its state society membership the first year unified membership was operative and *lost* 500 members from their state society the first year it was removed. The decline in both AMA and New York State Society membership has continued at an alarming rate since 1970; and, certainly proves that the problem isn't unified membership.

The real culprit is the frustration that we all feel more and more from governmental intrusion and intervention in our professional lives. The handiest kicking boy for many physicians is organized medicine, which they feel somehow has let them down and allowed government to get away with all sorts of horrible things. They respond by withdrawing into their own shells, rather than joining or encouraging the fight against the real enemy — governmental intrusion. We have to impress upon our faint-hearted brethren that the best defense is a good offense and with numbers there is strength. As former Congressman Eugene Keogh said in a message to the AMA House of Delegates during the 1975 Annual Meeting, "An AMA unitied, with singleness of purpose is an unsurmountable force."

John F. Kahle, M.D.
President, ArMA

A Thank You

Herbert K. Abrams, M.D., has recently assumed the directorship of the newly-funded Educational Resource Center for Occupational Safety and Health at the University of Arizona. Although he will retire from the Headship of the Department of Family and Community Medicine in July, 1978, he will continue as a professor in his new capacity. The development of the new Educational Resource Center will focus increased attention on occupational medicine in the Department of Family and Community Medicine, the College of Medicine, and other units of the Health Sciences Center and University.

Soon after arriving in Tucson in 1968, Dr. Abrams undertook the establishment of the El Rio Santa Cruz Neighborhood Health Center of Tucson's west side. The Neighborhood Health Center, recently relocated into a new Ambulatory Care Facility, serves as a major multi-million dollar health care resource for Tucson.

In 1971, the Department of Family and Community Medicine was established, expanding the previous Department of Community Medicine. Dr. Abrams remained as Head of the new Department at the University of Arizona College of Medicine. Shortly thereafter, the residency program in Family Practice was established. The residencies under Dr. Abrams' leadership have provided graduate training to over seventy residents to date. Many of them are serving Arizona communities as family physicians and community medicine experts.

Dr. Abrams' leadership in the medical school has fostered outreach into the rural areas of Arizona as well. His Department has assisted several communities in developing health services and attracting physicians. The Benson Health Services and the Marana Community Clinic may be the most visible and well-established of these sites, but many communities attest to the aid and encouragement received whenever Dr. Abrams was contacted.

Under Dr. Abrams' leadership, the Department of Family and Community Medicine has extended its educational services beyond the medical students and residents at University Hospital. New modalities for obtaining continuing medical education have been successfully developed in an attempt to meet the wide diversity of needs. It is not surprising, therefore, that the next phase in Dr. Abrams' career will be the development of educational services for providers of occupational health and safety services to Arizona working people. If the growth of Family and Community Medicine in the College of Medicine is an indicator, the Educational Resource Center for Occupational Safety and Health will be a major operation in short order.

For all his past accomplishments and the impetus to develop new goals and new roles for the College of Medicine, we of the faculty and the people of Arizona are truly grateful.

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Dean, College of Medicine
and
Jesse W. Tapp, Jr., M.D.
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A View From The Other Side

The reasons given by trial lawyers for not changing the existing tort system to a "no fault" system are open to question. The principal contention is that malpractice actions are on the increase because physicians are increasingly guilty of malpractice. Let's take a look at that in terms of the situation thirty odd years ago, immediately after W.W.II when over thirty companies wrote malpractice policies in Arizona for all risks at a premium of under $100. Perhaps the bulk of surgery then was performed by doctors who were not Board certified and had little formal training which would enable them to handle an unexpected difficult surgical problem. Virtually no patient sued the doctor in those situations which today would be uncontested liabilities. The disease was blamed for the bad result, not the doctor. How then can it be said that malpractice is increasing? The fact is that there is less malpractice today than ever before. It is the exposure that is on the increase, and this exposure is the result of legal doctrines that apply concepts of an absolute warranty of wholesomeness and safety in all situations. We haven't seen the statement that the reason more manufacturers are being sued is because their products are poorer, or that more architects and engineers are being sued because they are building poorer buildings and bridges. The latter group have become target defendants also, although they are better trained and have better materials to work with than ever before, just like physicians.

The next contention on the part of the lawyers is that the public interest is served by a judgment of negligence in a malpractice action as the only way to deter physicians from negligent acts, since physicians' groups and state agencies seldom invoke disciplinary action against doctors. It is difficult to visualize plaintiff lawyers in the role of crusaders for better medicine by way of this circuitous route. The facts are that when disciplinary action is taken against a doctor, he hires a lawyer and the disciplinary body frequently discovers that they have trampled on his client's rights. There is an instance in this state where the court that convicted a doctor of a felony, (statutory rape) ordered the Board of Medical Examiners to restore his license. What we need to hear from the lawyers is *how* to clean our house. How much of a contribution to the public interest is the tremendous increase in the cost of medical care brought about by the necessity of viewing each patient as a potential adversary?

It is suggested by the lawyers that malpractice premiums are not too high in relation to total income earned by physicians. An $800,000.00 attorney's fee as a result of a recent judgment in this state isn't exactly pocket change. It would take a physician many years of heart ache, tension, sleepless nights and travail before he could earn such a sum. It is submitted that if physicians' fees were reduced by half, there would be no reduction in premiums. There will be no reduction in malpractice suits or awards either until the public discovers that it is a mistake to indemnify themselves against medical accidents by requiring the medical profession to pool resources for that purpose.

Paul B. Jarrett, M.D.

REFERENCE

"Overview of the Medical Malpractice Dilemma." Alex Naimon, Federal Bar News, Vol. 24, No. 1, Jan. 1977.

The Arizona History Project And Continuing Medical Education In Arizona

The Project is trying to secure data on the history of group projects which were undertaken by local physicians in different communities in order to keep themselves abreast of medical changes.

Essays and orations were prepared for meetings of the Arizona Medical Association early in its meetings. Some of these papers were prepared by local physicians. Others were invited guests. And of course this has been continued all through the years and is more elaborate and covers a wide range of subjects at the present time.

But we're more interested in what happened locally. We have some inkling of some of these occurrances. For instance, Dr. James R. Moore states that in 1919 after he returned from W.W.I and was practicing at Jerome, Arizona he used to make monthly trips to Prescott over Mingus Mountain, a thirty-five mile relatively unimproved road, in order to attend a clinical discussion group which met at the Whipple Veteran's Hospital. You may recall that at that time by Presidential fiat, the United States Public Health Service was directed to take care of the returning veterans from World War One. The Whipple Hospital, previously an Army installation at Fort Whipple, was taken over by the Public Health Service and a great many of the physicians who served in those early days were returning veterans themselves.

At any rate, Dr. Moore describes very clearly that this was an early "continuing education" program carried on by the Yavapai County Medical Society.

Other early notes from the Maricopa County Medical Society indicate that they met once a month on Saturday night at the late lamented famous Chinese restaurant on Central Avenue, where they engaged in discussion of assigned subjects. Members were to prepare an essay on their subjects and present it for discussion. This continued apparently for two or three years.

So far as Phoenix is concerned, probably the earliest such organized club was the Phoenix Clinical Club. But there is no clear data so far to establish exactly when it began. It is known that W. Warner Watkins, M.D., a pioneer radiologist-pathologist, was one of the moving forces. But exactly who the original members were and exactly when it began we have not been able to ascertain.

We should certainly like to hear from all of the luncheon clubs that are now functioning over the state and I am sad to relate that we have no data on the formation of such clubs in Tucson and I am sure that they were no laggards in this activity.

This is an appeal for information concerning these varied and wide-spread state activities that doctors used in times gone by to keep themselves abreast of medical progress. Some of the more recent practitioners of the art seem to feel that they have discovered a whole new and previously unknown method of self-education. It seems to some of us that the main thing that is new is the horrendous cost of registration.

John W. Kennedy, M.D.

Correspondence

Arizona Medicine
Dear Editor:

I must disagree with the position taken by the Arizona Medical Association in opposing the passage of a law enabling optometrists to write for certain prescription drugs.

I have several reasons for disagreeing with our official position. Most importantly it is inconsistent with our more generally held opinion that the government is interfering too much already in the practice of medicine.

My first contention is that while the government can encourage, it cannot *insure* the quality of anything — let alone the quality of medical care. The pre-eminence and excellence of the medical profession in this country was certainly not achieved — and will not be maintained — by legislative fiat. Most of us have been quite skeptical of government planners' efforts to "improve the quality of medical care." In most cases where physicians are practicing less than quality care, it is the medical community itself through both formal and informal self-imposed controls that restrain improper behavior. Although as an MD I am legally empowered to write prescriptions for disorders of the eye, I refuse to do so. As a urologist I do not feel competent in the field of ophthalmology. However, it is my own ethical standards that keep me from prescribing these drugs and not any arbitrary laws. Extending our argument to its logical conclusion, one would have to argue that optometrists know more about certain conditions of the eye than urologists and a law should be passed to restrict urologists as well as optometrists (now there is a *real* Pandora's Box).

My second argument is that our fears in many ways seem exaggerated or imagined. Now I am not a foolish person, and I know that there would be *some* unethical or uninformed optometrists who would try to overstep their abilities and do more than they are trained to do (just as we have *some* unethical or uninformed physicians who do the same). I know there would be some patients who would suffer at the hands of these less capable optometrists (just as we have patients who suffer at the hands of less capable MD's). But I cannot help but believe that *most* optometrists, like *most* physicians, are decent people and have to live with themselves when they go home at night: they would respect their own limitations as we respect ours. In those cases where an optometrist causes suffering and damage to a patient, he would be liable to be sued for malpractice as we MD's are. In the long-run, if any health care provider — be he medical doctor or optometrist — is inadequate he will eventually be judged by the forces of public opinion and free competition.

My last argument is the most abstract but most important of all. I contend that it is the *individual patient* who ought to have the ultimate right and responsibility for deciding what is good or bad for his own health in any given situation. Just as I, in my own practice, cannot force a patient to follow my advice against his will even when I believe it to be in his own best interests; so we as an organization should not presume to do so (through government) to the public at large. If some people are determined to seek care from optometrists, naturopaths or chiropractors, we must be prepared to explain the differences between them and us and then hope for the best. As soon as set up an agency (no matter how benevolent) through the coercive powers of the state as a watchdog, we displace the responsibility from the individual patient to that agency and finally to the society at large that funds that agency. Rights cannot be had without responsibilities. If government assumes the responsibility for health, then it also must assume the right to decide how health will be dispensed. This is exactly where we find ourselves in America today: trudging down that long and sorrowful road that leads to increased government planning, spending, regulation and ultimately socialism, with the attendant loss of individual liberty that necessarily accompanies that latter unhappy collectivist state.

"What s good for the goose is also good for the gander." There is no compelling reason why in ten years time the government will not also be able to claim — in the interests of public welfare of course — that general practitioners cannot perform surgery or obtain hospital privileges or that urologists are only allowed to write prescriptions for one class of drugs that pertain to diseases of the urinary tract. We in the medical profession have been inadvertently encouraging government to assume those awesome powers for several decades. We are paying dearly for it now with our freedom. "We have met the enemy, and they are us."

This is a marvelous opportunity for ARMA to help to begin to reverse the tide of paternalism and welfarism that is strangling our nation. It will take courage on our part to support such an unpopular cause. We will have to reeducate the public and ourselves. It will make us uncomfortable for awhile and I do not deny that there is potential danger that excesses will, in fact, be perpetrated by the unscrupulous. However, I am fully confident that these excesses, given time, can be controlled in ways that are still consistent with high quality medical care and the private practice of medicine. Either we truly believe in the principles of the free market, private practice, true competition and free choice of health care providers and will defend that system even when it is not to our perceived advantage, or we will otherwise simply lend credence to the arguments of our opponents who claim that free enterprise cannot work and want to replace it with a regulated, controlled, and socialistic economy. History teaches us that there can be no middle ground. One of the sacrifices we will have to make will be to give up our virtual monopoly in providing health care to patients. If we do not do it now voluntarily and intelligently, bureaucrats will do it for us anyway when American medicine is controlled by a federal health planning czar. Most horribly, we ourselves will have been the unwitting accomplices in the destruction of what is generally agreed to be the finest health care system in the world.

Sincerely yours,

Murray S. Feldstein, M.D.

Errata

Inadvertently the references to the article "Clinical Depression Complicating Chronic Disease" by Stephen B. Shanfield, M.D. were omitted. This appeared in the Emotional Disorders Section of the January 1978 issue of *Arizona Medicine*. These references can be obtained by contacting the offices of *Arizona Medicine*.

of the circulatory system,[14] may raise some questions about the use of these agents to treat a disease that is primarily cosmetic. The increased mortality in oral contraceptive users appears most marked in older patients and in patients who smoke cigarettes. Hirsute patients having an ovarian source for their excess androgens may want to consider simple, mechanical methods of hair removal such as shaving, plucking, waxing, bleaching, chemical depilation or electrolysis.

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ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

ARTICLES OF INCORPORATION AND BYLAWS COMMITTEE

Meeting of the Articles of Incorporation and Bylaws Committee of the Arizona Medical Association, Inc., held on Saturday, November 12, 1977 at 810 West Bethany Home Road, Phoenix, Arizona, convened at 2:08 p.m., Charles E. Henderson, M.D., Chairman, presiding.

COMMITTEE MEMBERSHIP

The Committee considered request by the Board of Directors to review the general problem as expressed by the Pima County Medical Society and James E. Brady, M.D., of appointments of non-member physicians to committees of the Association. Mr. Jacobson, General Council for the Association, responded to an inquiry "may physicians who are not members of ArMA be appointed as regular members of ArMA Committees?" by stating in part, that "assuming, with respect to the committee in question, that (a) there is neither a bylaw prohibition, expressed or implied, nor (b) does the committee do more than recommend to the Board, the answer is 'yes'." There was considerable discussion on the pros and cons of non-member physicians being appointed to committees of the Association.

IT WAS MOVED AND CARRIED TO INFORM THE BOARD OF DIRECTORS THAT THE ARTICLES OF INCORPORATION AND BYLAWS COMMITTEE HAVE CONSIDERED THE PROBLEM OF APPOINTMENT OF NON-MEMBER PHYSICIANS TO COMMITTEES OF THE ASSOCIATION EXTENSIVELY AND THAT IT IS RECOMMENDED THAT NO CHANGES BE MADE AT THE CURRENT TIME. IT IS FURTHER RECOMMENDED, HOWEVER, THAT ALL COMMITTEE APPOINTMENTS SHOULD BE CAREFULLY SCRUTINIZED BY THE BOARD OF DIRECTORS.

GEOGRAPHIC DISTRIBUTION AND ELECTION OF DISTRICT DIRECTORS—RESOLUTION #A-3-77

Resolution #A-3-77 as considered by the

House of Delegates during the 1977 annual meeting and referred to the President for study, was considered by the Committee as requested by Dr. Kahle.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE FOLLOWING RESOLUTION BE INTRODUCED TO THE HOUSE OF DELEGATES DURING THE NEXT ANNUAL MEETING:

RESOLVED, That Chapter V, Section 6, be amended to read as follows:

Section 6. Geographic Distribution and Election of District Directors: There shall be six state districts which shall be designated as Central (Maricopa County); Northeastern (Apache, Gila and Navajo Counties); Northwestern (Coconino, Mohave and Yavapai Counties); Southeastern (Cochise, Graham, Greenlee and Santa Cruz Counties); Southern (Pima County); and Southwestern (Pinal and Yuma Counties): [*Four of the districts, Northeastern, Southeastern, Southwestern shall have one Director elected from each; the Southern district shall have three Directors; and the Central district shall have five Directors*] EACH DISTRICT SHALL BE REPRESENTED BY ONE DIRECTOR FOR THE FIRST 200 ACTIVE MEMBERS OR FRACTION THEREOF: AND ONE DIRECTOR FOR EACH ADDITIONAL 200 ACTIVE MEMBERS OR FRACTION THEREOF, AS DETERMINED ON OCTOBER FIRST PRECEDING THE ANNUAL MEETING. Terms of District Directors shall not run concurrently.

NOTE: All bracketed, italicized words represent deletions; all fully capitalized words are new material.

PHYSICIAN'S ASSISTANTS MEMBERSHIP CATEGORY

The Board of Directors requested that the committee consider a membership category for Physician's Assistants and develop resolution for consideration by the House of Delegates during its next regular meeting.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE FOLLOWING RESOLUTIONS BE INTRODUCED TO THE HOUSE OF DELEGATES DURING THE NEXT ANNUAL MEETING:

RESOLVED, That Chapter 11, Section 3(E) be amended to read as follows:

(E) Affiliate Members. Affiliate members may be elected to this Association by the Board upon recommendation by county societies from (1) those doctors of medicine who are duly accredited in Mexico, or in foreign countries, engaged in medical missionary and similar educational philanthropic work, (2) Arizona dentists who are members in good standing of their local and state societies, (3) pharmacists who are active members of their Arizona State Association, (4) teachers of medicine and allied sciences who are not eligible to other membership in this Association, (5) former active members of this Association, who are now in practice in another state, [*and*] (6) retired physicians holding degree of Doctor of Medicine and residing in Arizona, AND (7) THOSE PERSONS CERTIFIED AS PHYSICIAN'S ASSISTANTS AND RESIDING IN ARIZONA. Rights. Affiliate members shall enjoy the privileges of attending the scientific meetings. They shall not have the right to serve as Delegates, or to hold elective office. They shall pay no dues and not be entitled to receive any publication of the Arizona Medical Association except by subscription.

NOTE: All bracketed, italicized words represent deletions; all fully capitalized words are new material.

BYLAW AMENDMENTS REGARDING SECTIONS OF THE PROFESSIONAL COMMITTEE

The Board of Directors during meeting held September 24, 1977 requested the committee to consider Bylaw Amendments to provide for a section on Mental Health and Substance Abuse under the Professional Committee while striking the current separate sections on Drug Abuse and Mental Health.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE FOLLOWING RESOLUTION BE INTRODUCED TO THE HOUSE OF DELEGATES DURING ITS NEXT ANNUAL MEETING:

RESOLVED, That Chapter VII, Section 4 (n) be amended to read as follows:

(n) Professional: —This committee shall consist of a Chairman and at least ten members. A Chairman shall be appointed annually from among the members who have served for at least three years on the committee.

The committee will be responsible for all professional aspects of medicine, other than the economics and legislative areas and those areas covered by other standing committees, and comprise sections on Aging and General Medicine; Allied medical groups; Athletic medicine; Disaster medicine; [*Drug abuse;*] Emergency Care; Medicine and Religion; Mental health AND SUBSTANCE ABUSE; Poison control; Public health; Rehabilitation Medicine; Safety and such other sections as may be indicated from time to time. It shall interest itself in medico-legal problems. Further, an advisor to the Woman's Auxiliary of ArMA shall be designated (to be chosen from the same geographic area of the state as the current President of the Woman's Auxiliary).

Each committee member, except the Chairman, shall serve as Chairman of a section for a particular activity. The Chairman may also serve as the Chairman of a section. The section Chairman shall be appointed by the President of the Association, with advice of the Chairman of the Professional Committee.

NOTE: All bracketed, italicized words represent deletions; all fully capitalized words are new material.

MATERNAL AND CHILD HEALTH CARE COMMITTEE

The meeting of the Maternal and Child Health Care Committee of the Arizona Medical Association, Inc., held Saturday, December 10, 1977, at 810 West Bethany Home Road, Phoenix, Arizona, convened at 1:28 p.m., William C. Scott, M.D., Chairman, presiding.

BIRTH DEFECTS REGISTRY

It was pointed out to the committee that coordination of the data systems of the Arizona Perinatal Program and of the Maternal and Child Health Bureau of The Department of Health Services (through the Arizona State Newborn Transport and Intensive Care Program) could contribute rapidly to the development of a birth defect registry for Arizona alone or in association with the CDC in Atlanta and/or Ann Arbor, Michigan. Every effort should be made to seek uniform registry of such defects among *newborn and still-born* infants in Arizona.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE ASSOCIATION SUPPORT THE ESTABLISHMENT OF A BIRTH DEFECTS REGISTRY FOR ARIZONA.

It was understood that this support would not commit financial resources of ArMA.

ARIZONA PERINATAL PROGRAM DATA SYSTEM

The Arizona Perinatal Program data system is currently being successfully used in all institutions under contract to that program. Having this system expanded to all hospitals in Arizona (particularly Tucson Medical Center, Desert Samaritan Hospital, and the Phoenix Indian Medical Center) would markedly expand and enhance that data base.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE ASSOCIATION ENDORSE THE EXTENSION OF THE ARIZONA PERINATAL PROGRAM DATA SYSTEM TO ALL HOSPITALS IN THE STATE AND THAT AN EXTENSIVE PHYSICIAN EDUCATION PROGRAM OF THE DATA SYSTEM BE SUPPORTED.

LEGAL LIABILITY OF MIDWIFERY REGULATIONS

The Department of Health Services is about to issue rules regarding the licensing of midwifery. The committee was concerned about certain aspects of the rules which may place the physician into a legal liability situation.

IT WAS MOVED AND CARRIED TO ASK THE BOARD OF DIRECTORS FOR PERMISSION TO HAVE THE ASSOCIATION'S LEGAL COUNSEL REVIEW THESE PROPOSED RULES TO DETERMINE WHAT IMPOSITION OF LIABILITY, IF ANY, THAT THESE RULES IMPOSE ON PHYSICIANS.

GENETICS LABORATORY

The question of whether to recommend that it would be efficacious to dominantly utilize the genetics laboratory at the University of Arizona in Tucson was discussed at some length.

IT WAS MOVED AND CARRIED TO REFER THIS MATTER BACK TO THE SECTION ON PERINATAL HEALTH FOR FURTHER STUDY, SUCH STUDY TO INCLUDE CONSULTATIONS WITH PHOENIX LABORATORIES AS WELL AS PRIVATE LABORATORIES IN TUCSON.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc., held at 4622 N. Seventh Avenue, Phoenix, Arizona on Friday, December 16, 1977, convened at 7:10 p.m., John F. Kahle, M.D., President and Chairman, presiding.

RATE REVIEW OF PROFESSIONAL FEES

Mr. Jacobson reported that little has changed on this matter since last reviewed. We are still awaiting final settlement and agreement between the attorneys and the Department of Health Services—RECEIVED.

AMERICAN MEDICAL WOMEN'S ASSOCIATION

Andrew M. Goldner, Ph.D.'s letter of November 29, 1977 expressing appreciation for our assistance in sending Ms. Susan Tharp to the subject meeting held in Colorado was received.

REVIEW OF BOARD OF DIRECTORS AGENDA

The agenda for the December 17, 1977 meeting of the Board of Directors was reviewed with comments and suggestions made.

SCIENTIFIC ASSEMBLY COMMITTEE

The meeting of the Scientific Assembly Committee of the Arizona Medical Association, Inc., held in the Sinaloa Room, Marriott Hotel, 180 West Broadway, Tucson, Arizona, on Saturday, December 3, 1977, convened at 1:05 p.m., Richard A. Silver, M.D., Chairman, presiding.

CALIFORNIA COLLEGE OF MEDICINE

The committee reviewed the final program as presented by the California College of Medicine faculty and made no changes.

LOCAL PAPERS

IT WAS MOVED AND CARRIED TO APPROVE THE FOLLOWING PAPERS SUBMITTED BY ARIZONA PHYSICIANS:

1. Space Age Diagnosis of Gallbladder Disease—M. Herbert Nathan, M.D.
2. Spectrum of Thyrotoxicosis: New Varieties Which Alter Therapy—Marshall B. Block, M.D.
3. Multiple Sclerosis in Arizona: A Clinical Profile—Colin R. Bamford, M.D.
4. Rehabilitation of the Stroke Patient—Stuart I. Holtzman, M.D.
5. Initial Management of Head Injuries—Philip R. Weinstein, M.D.
6. Differential Diagnosis Including Case Presentations of Tuberculosis—Laurence Farer, M.D.
7. Perioperative Myocardial Infarction in Aortocoronary Bypass Surgery—Charles M. Rucker, M.D.
8. The Adult Minimal Brain Dysfunction: The Diagnostic Problem—Harris D. Murley, M.D.
9. Medical Care in the Rural Communities in the State of Arizona—Dermont W. Melick, M.D.
10. Current Status of Diagnosis and Treatment of Carcinoma of the Prostrate—Thomas H. Stanisic, M.D.
11. Evaluation of Male Infertility—Mark A. Silvert, M.D.
12. Renal Transplantation in Children: The Phoenix Experience—Karen C. Cartwright, M.D.
13. Early Rehabilitation and Return to Function Following Rigid Internal and External Fixation of Bone Using the AO (Swiss) Technique—Saul N. Schreiber, M.D.
14. Rotational Subluxation of the Carpal Navicular and Intercillary Instability of the Wrist: A Frequently Missed Roentgen Diagnosis—John C. Bjelland, M.D.
15. An Analysis of Differential Spinal Anesthesia and Pentathol Pain Study in the Differential Diagnosis of Back Pain—John F. Krempen, M.D.
16. The Distressed Physician: Where Do We Go From Here?—Thomas E. Bittker, M.D., Stephen C. Scheiber, M.D., and Emanuel M. Steindler.

SCIENTIFIC EXHIBITS

IT WAS MOVED AND CARRIED TO APPROVE THE FOLLOWING EXHIBIT APPLICATIONS:

1. Non-Invasive Testing of the Peripheral Vascular System—John M. Johnson, M.D.
2. Disability Evaluation Under Social Security—Vernon Kores, M.D.
3. Exercise and Preventive Cardiology—Howard J. Reuben, M.D.
4. Clinical Experience With Hoffmann External Fixation Device—Joseph J. Williams, M.D.
5. Pediatric Hypertension Clinic and Outreach Programs—Audrey Rath, R.N.
6. Rotational Subluxation of the Carpal Navicular and Intercillary Instability of the Wrist: A Frequently Missed Roentgen Diagnosis—John C. Bjelland, M.D.
7. Acute Bowing Fractures of the Extremities: A Frequently Missed Roentgen Diagnosis—John C. Bjelland, M.D.
8. Automobile Safety for the Infant and Child—C.W. Pullen, M.D.
9. Replantation and Microsurgery—Ronald B. Joseph, M.D.
10. The "Battered Child" Syndrome—Alex Newman, M.D.

IT WAS MOVED AND CARRIED TO ACCEPT AN EXHIBIT OF "HOME HEALTH SERVICES IN ARIZONA" BY DOROTHY E. GERRARD, R.N., IF A SPACE SHOULD BE AVAILABLE IN THE COMMERCIAL EXHIBIT AREA.

It was also agreed that an exhibit on ambulatory surgical facilities proposed by Boyden Crouch, M.D., would be accepted should a second commercial exhibit space be available.

COMPUTER ASSISTED INSTRUCTION

The committee approved the following topics for the subject program:

Multiple Choice Format
- Title
 - CARDIOPULMONARY RESUSCITATION
 - HYPERTENSIVE EMERGENCIES
 - HYPERTENSION MANAGEMENT

Vocabulary Format
- Title
 - COMA
 - GI BLEEDING
 - HYPERTENSION DIAGNOSIS
 - JAUNDICE
 - JOINT PAIN
 - TRAUMA

Natural Language Format
- Title
 - FLUID AND ELECTROLYTE MANAGEMENT

Combination or Other Input Format
- Title
 - RESPIRATORY SIMULATION

REORGANIZATION OF FORMAT OF STATE NURSING

A suggestion to organize the Annual Meeting so that State Specialty Societies would hold their annual meetings concurrently and programs be developed to meet needs of the various specialties was discussed and tabled indefinitely, pending further investigation, as follows:

IT WAS MOVED AND CARRIED TO REQUEST THE EXECUTIVE DIRECTOR TO CONTACT THE GEORGIA MEDICAL SOCIETY FOR INFORMATION REGARDING THE FORMAT OF THEIR ANNUAL MEETING AND SUBSEQUENTLY TO CONTACT ARIZONA SPECIALTY SOCIETIES TO SEEK THEIR REACTION TO HOLDING THEIR ANNUAL MEETINGS AT THE TIME OF THE ArMA ANNUAL MEETING.

AD HOC COMMITTEE ON THE U.S. SUPREME COURT DECISION REGARDING ADVERTISING

The meeting of the Ad Hoc Committee on the U.S. Supreme Court Decision Regarding Advertising of the Arizona Medical Association, Inc. held Saturday, January 14, 1978 at 810 W. Bethany Home Road, Phoenix, Arizona convened at 11:02 a.m., Richard L. Dexter, M.D., President-Elect and Chairman, presiding.

REVIEW

Mr. Jacobson reviewed his letter of November 29, 1977 (copy provided to all members of the committee with enclosures). Mr. Jacobson explained that "the committee now has before it four versions of a proposed statute representing three approaches to the problem, all of which are legal. A judgemental decision as to which to select is not in order. The sooner it is made, the better. That way Gary Barnett will have enough time to begin to prepare the ground work for introduction and passage.

The committee will be faced with the following choices:

1. A statute which lists only activities declared illegal by the U.S. Supreme Court.
2. A statute which lists activities declared illegal, activities declared legal and the scope of possible rule-making power, all as set forth by the U.S. Supreme Court.
3. Two versions of a statute which list some but not all of the activities specifically declared legal by the U.S. Supreme Court but otherwise include all the material in number 2 above."

Mr. Buri's recommendation, listing only activities declared illegal, received the greatest attention of the committee. This approach would require the Medical Practice Act to be amended to read "advertising in a false, deceptive or misleading manner, advertising the quality of medical services, advertising by in-person solicitation, and advertising procedures and practices which are illegal instead of "Advertising" as unprofessional conduct.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE LEGISLATIVE COMMITTEE AND THE BOARD OF DIRECTORS THAT THE ASSOCIATION PROPOSE LEGISLATION DURING THIS SESSION OF THE LEGISLATURE TO AMEND THE MEDICAL PRACTICE ACT BY PROVIDING FOR A NEW DEFINITION OF ADVERTISING UNDER UNPROFESSIONAL CONDUCT TO READ "ADVERTISING A FALSE, DECEPTIVE OR MISLEADING MANNER, ADVERTISING THE QUALITY OF MEDICAL SERVICES, ADVERTISING BY IN-PERSON SOLICITATION, AND ADVERTISING PROCEDURES AND PRACTICES WHICH ARE ILLEGAL."

It was determined that Dr. Dexter would seek the approval of the committee's recommendation from the Legislative Committee and the Executive Committee during its next meeting in order to prepare the ground work for introduction and passage of this legislation on a timely basis.

HEALTH MANPOWER COMMITTEE

Meeting of the Health Manpower Committee of the Arizona Medical Association, Inc., held Wednesday, January 11, 1978, at 810 W. Bethany Home Road, Phoenix, convened at 7:30 p.m., Louis C. Kossuth, M.D., Chairman, presiding.

INFORMATION ITEMS PHYSICIAN ASSISTANT MEMBERSHIP IN ArMA

Dr. Kossuth reported that the Board of Directors had referred the recommendation that Physician Assistants be accepted into ArMA Membership to the Articles of Incorporation and Bylaws Committee. A Bylaws Amendment will be introduced to the House of Delegates in April, 1978, which if passed would open Affiliate Membership to Physician Assistants.

LETTERS TO COUNTY MEDICAL SOCIETIES

Only two replies were received to letters sent to all county medical societies—one from Mohave County stating they felt there were no communities in their county in need of physicians, and one from Cochise County listing several communities where they felt one or more physicians are needed.

ARCOSANTI FESTIVAL

Dr. Kossuth called attention to an article in the *Arizona Republic* reporting on the above. General discussion ensued. For information only.

ARIZONA RURAL HEALTH CONFERENCE

Dr. Vuturo presented the program and budget for the Seventh Annual Arizona Rural Health Conference. Date is Friday and Saturday, May 12-13, 1978. This year's conference will include Rural Health and Emergency Care. One day's program is entitled "Emergency Health Planning and Delivery: Realistic Costs and Mechanisms for Cost Control." The other day will be devoted to "Financing Rural Health Care."

After review of the program and the budget, the committee took the following action:

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE ARIZONA MEDICAL ASSOCIATION, INC. CONTRIBUTE $1,000 TO THE SEVENTH ANNUAL ARIZONA RURAL HEALTH CONFERENCE SCHEDULED FOR MAY 12-13, 1978.

Dr. Vuturo indicated he will also seek financial support from the Health Systems Agencies, as suggested by Dr. Kossuth.

PHYSICIAN'S ASSISTANTS REGULATIONS ADOPTED BY THE JOINT BOARD

The regulations recently adopted by the Joint Board of Medical Examiners and Osteopathic Examiners in Medicine and Surgery relating to Physician's Assistants were reviewed by the committee.

IT WAS MOVED AND CARRIED TO FORWARD THE REGULATIONS ON PHYSICIAN'S ASSISTANTS TO THE BOARD OF DIRECTORS WITH THE RECOMMENDATION THAT THEY TAKE A FAVORABLE POSITION REGARDING THE REGULATIONS.

FUNDING FOR ADMINISTRATION BY THE JOINT BOARD

Letter dated 12/23/77 from the Chairman of the Joint Board outlining the problems of lack of funding for the Joint Board to carry out administration of the Physician's Assistants program was reviewed by the committee.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT ArMA SUPPORT THE NECESSITY FOR FUNDING FOR THE JOINT BOARD OF MEDICAL EXAMINERS AND OSTEOPATHIC EXAMINERS IN MEDICINE AND SURGERY IN ORDER THAT THEY MAY CARRY OUT THE OPERATION OF THE PHYSICIAN'S ASSISTANTS PROGRAM: AND THAT ArMA RECOMMEND TO THE LEGISLATURE APPROVAL OF A SUPPLEMENTAL BUDGET REQUEST BY THE JOINT BOARD.

PHYSICIAN EXTENDERS H.R. 84-22

Dr. Kossuth read from portions of the above bill passed by Congress on November 30, 1977, concerning payment by Medicare and Medicaid of fees for services of physician extenders. For information.

ARIZONA HOUSE BILL 2003

The above bill, distributed to committee members for study prior to the meeting, was discussed. Members expressed strongly their feeling that the legislation must be opposed, and took the following action:

IT WAS MOVED AND CARRIED TO RECOMMEND THAT THE ARIZONA MEDICAL ASSOCIATION, INC. ACTIVELY OPPOSE HOUSE BILL 2003 TO THE UTMOST EFFORT THAT CAN BE EXERTED.

Other documents presented to the committee, including copies of resolutions adopted at the Biennial Convention of the Arizona Nurses Association on November 5, 1977, concerning Nurse Practitioners were reviewed by the committee.

SINGLE BOARD FOR PHYSICIAN EXTENDERS

The previous recommendation of this committee that *all* physician extenders be placed under one certifying board was returned to the committee by the Board of Directors for further clarification. After discussion, the following action resulted:

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT ArMA PROPOSE LEGISLATION TO PLACE ALL PHYSICIAN'S ASSISTANTS AND NURSE PRACTITIONERS IN THE EXTENDED ROLE, REGARDLESS OF THE TITLE THEY MIGHT USE, UNDER THE JURISDICTION OF THE JOINT BOARD OF MEDICAL EXAMINERS AND OSTEOPATHIC EXAMINERS IN MEDICINE AND SURGERY.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc., held at 810 W. Bethany Home Road, Phoenix, Arizona, on Saturday, December 17, 1977, a quorum being present, convened at 10 o'clock a.m., John F. Kahle, M.D., President and Chairman, presiding.

ArMPAC REPORT

James A. Austin, M.D., reported that as of this date (December 17, 1977), ArMPAC had already received 140 memberships for the 1978 dues year. He attributed this success to the reinstitution of unified membership billing by the Maricopa and Pima County Medical Societies. It is ArMPAC's goal for 1978 to reach 1,000 members by the end of the year. Dr. Austin urged that all members of the Board of Directors become sustaining members of ArMPAC. He also advised that Senator Dennis DeConcini had been invited to be the guest speaker at the annual ArMPAC banquet scheduled for Thursday, April 27, 1978 during the 1978 Annual Meeting in Tucson.

LEGAL FEES

Discussion ensued over legal fees incurred as a result of Federal Election Commission rules and regulations.

IT WAS MOVED AND CARRIED TO REFER THE MATTER OF THE LEGAL FEES TO THE FINANCE COMMITTEE FOR THEIR RECOMMENDATION AS TO THE DISTRIBUTION OF THIS EXPENSE WITH FURTHER REFERRAL TO THE EXECUTIVE COMMITTEE FOR PAYMENT.

AUXILIARY REPORT

Maude O. Reno, President, reported as

follows:

Community Health and Family Health Committee:

The immunization awareness program using the hop scotch stencil to paint grade school playgrounds has been carried out in Yavapai and Maricopa Counties. The other counties either could not get volunteers to do this or were refused permission by the schools. The Heimlich maneuver is not on the milk cartons as yet. A letter was sent recently to the dairy companies to remind them of our request.

Health Education:

Health career charts are being distributed in the high schools. Hamer Education Loan Funds, loans to college students in the health field, are not requested as much as in the past several years.

Membership:

This year an extensive effort is toward reaching the wives of physicians in unorganized Auxiliary counties. We need the home addresses and the spouse's first name. We have found that the young wife is either in a career or furthering her education. She is not as inclined to do volunteer work.

Communications:

Publicity on Auxiliary's grant to the University of Arizona Medical School students for loans, Owens Associates have had difficulty to get newspapers to publish this article and picture. The Family Health committee has a newsletter to all county presidents titled "Pacemaker". They have made several suggestions which include Action for Children's Television; Bike Rodeo which is a safety education; "Mr. Ugh" labels available that warn of poisonous contents within, and to conserve energy and have fewer burn threats by turning the water heater down to 130 degrees.

National Conference:

Four county presidents-elect, Arizona president and president-elect attended a three-day conference in Chicago in October. There were eight seminars. Transportation was paid by national for the county presidents-elect. This was a leadership and educational conference.

Long Range Planning Committee:

This committee is studying the problems of Auxiliary and will make recommendations in February. Included in the study are retaining members; improving the fall workshop; survey of officers and chairmen's duties; survey of the Hamer Loan Fund—continue this or not to continue the assessment, and the possibility of obtaining a part-time employee.

State and County:

A workshop was held in November at the ArMA building. Forty-two attended; reports were given from the conference in Chicago and several state chairmen reported. Legislation was stressed.

The state president and president-elect have visited Yavapai, Coconino, Yuma and Maricopa Counties. All counties are active in many areas and busy raising funds for ERF. Yuma County members have taken the CPR courses but need an "Annie" which they are unable to borrow. Maricopa County has raised funds for a mobile unit to transport wheelchair and bed patients. Coconino County has had a bazaar for the crisis nursery and a local mission and ERF.

The Board received for information from Mrs. James L. Parsons, President-Elect. a letter of appreciation for ArMA's contribution to their activities as well as providing us with a budget and program for outline for 1978.

ARIZONA HEALTH SERVICE CORPS

Ward B. Studt, M.D., reported briefly on HEW's rejection of our proposal for second year funding for the proposed Arizona Health Service Corps.

A letter acknowledging the rejection of the proposal was received by the Board along with a copy of a letter to the Executive Committee of the Arizona Health Service Corps thanking them for their assistance in this project.

ARTICLES OF INCORPORATION AND BYLAWS COMMITTEE

Non-member Physicians on Committees

The committee recommended that no changes by made in the bylaws at the present time regarding the appointment of non-member physicians to ArMA committees—RECEIVED.

MEMBERSHIP OF PHYSICIAN'S ASSISTANTS

IT WAS MOVED AND CARRIED TO APPROVE THE INTRODUCTION OF THE FOLLOWING RESOLUTION IN THE 1978 HOUSE OF DELEGATES.

Subject: MEMBERSHIP OF PHYSICIAN'S ASSISTANTS IN THE ARIZONA MEDICAL ASSOCIATION

RESOLVED, That Chapter 11, Section 3(E) be amended to read as follows:

(E) Affiliate Members. Affiliate members may be elected to this Association by the Board upon recommendation by county societies from (1) those doctors of medicine who are duly accredited in Mexico, or in foreign countries, engaged in medical missionary and similar educational philanthropic work, (2) Arizona dentists who are members in good standing of their local and state societies, (3) pharmacists who are active members of their Arizona State Association, (4) teachers of medicine and allied sciences who are not eligible to other membership in this Association, (5) former active members of this Association, who are now in practice in another state, [*and*] (6) retired physicians holding degree of Doctor of Medicine and residing in Arizona, AND (7) THOSE PERSONS CERTIFIED AS PHYSICIAN'S ASSISTANTS AND RESIDING IN ARIZONA.

Rights. Affiliate members shall enjoy the privileges of attending the scientific meetings. They shall not have the right to serve as Delegates, or to hold elective office. They shall pay no dues and not be entitled to receive any publication of the Arizona Medical Association except by subscription.

NOTE: All bracketed, italicized words represent deletions; all fully capitalized words are new material.

DISTRICT DIRECTORS COMPOSITION

IT WAS MOVED AND CARRIED TO APPROVE THE INTRODUCTION OF THE FOLLOWING RESOLUTION IN THE 1978 HOUSE OF DELEGATES.

Subject: DISTRICT DIRECTORS—BOARD OF DIRECTORS COMPOSITION

RESOLVED, That Chapter V, Section 6, be amended to read as follows:

Section 6. Geographic Distribution and Election of District Directors: There shall be six state districts which shall be designated as Central (Maricopa County); Northeastern (Apache, Gila and Navajo Counties); Northwestern (Coconino, Mohave and Yavapai Counties); Southeastern (Cochise, Graham, Greenlee and Santa Cruz Counties); Southern (Pima County); and Southwestern (Pinal and Yuma Counties). [*Four of the districts, Northeastern, Northwestern, Southeastern, Southwestern shall have one Director elected from each; the Southern district shall have three Directors;*] *and Central district shall have five Directors.* EACH DISTRICT SHALL BE REPRESENTED BY ONE DIRECTOR FOR THE FIRST 200 ACTIVE MEMBERS OR FRACTION THEREOF, AND ONE DIRECTOR FOR EACH ADDITIONAL 200 ACTIVE MEMBERS OR FRACTION THEREOF, AS DETERMINED ON OCTOBER FIRST PRECEDING THE ANNUAL MEETING. Terms of District Directors shall not run concurrently.

NOTE: All bracketed, italicized words represent deletions; all fully capitalized words are new material.

PROFESSIONAL COMMITTEE SECTION DESIGNATION

IT WAS MOVED AND CARRIED TO APPROVE THE INTRODUCTION OF THE FOLLOWING RESOLUTION IN THE 1978 HOUSE OF DELEGATES.

Subject: PROFESSIONAL COMMITTEE SECTION DESIGNATION

RESOLVED, That Chapter VII, Section 4(n) be amended to read as follows:

(n) Professional —This committee shall consist of a Chairman and at least ten members. A Chairman shall be appointed annually from among the members who have served for at least three years on the committee.

The committee will be responsible for all professional aspects of medicine, other than the economics and legislative areas and those areas covered by other standing committees, and comprise sections on Aging and General Medicine; Allied medical groups; Athletic medicine; Disaster medicine; [*Drug abuse;*] Emergency Care; Medicine and Religion; Mental health AND SUBSTANCE ABUSE; Poison control; Public health; Rehabilitation medicine; Safety and such other sections as may be indicated from time to time. It shall interest itself in medico-legal problems. Further, an advisor to the [*Woman's Auxiliary of ArMA*] ARIZONA MEDICAL ASSOCIATION AUXILIARY, INC. shall be designated (to be chosen from the same geographic area of the state as the current President of the [*Woman's*] Auxiliary).

Each committee member, except the Chairman, shall serve as Chairman of a section for a particular activity. The Chairman may also serve as the Chairman of a

section. The section Chairman shall be appointed by the President of the Association, with advice of the Chairman of the Professional Committee.

NOTE: All bracketed, italicized words represent deletions; all fully capitalized words are new material.

BENEVOLENT AND LOAN FUND COMMITTEE

The letter from Jose Duncan expressing appreciation for receiving the scholarship was received for information.

BOMEX Communications

An exchange of correspondence between BOMEX and Richard S. Armstrong, M.D., regarding communications between the Board and hospital staffs was reviewed.

IT WAS MOVED AND CARRIED TO APPOINT A NEW COMMITTEE OR ASSIGN TO AN EXISTING COMMITTEE THE TASK OF WORKING WITH BOMEX AND THE ARIZONA HOSPITAL ASSOCIATION IN AN ATTEMPT TO SOLVE THE PROBLEMS EMANATING FROM BOMEX ACTIONS WHICH RESULT IN PHYSICIANS' HOSPITAL PRIVILEGES EITHER BEING DENIED OR LIMITED.

BOMEX Vacancy

It was reported that the term of Guy B. Atonna, M.D, on the Board of Medical Examiners expires on February 26, 1978.

Membership on AMA Councils

IT WAS MOVED AND CARRIED TO REFER THE REQUEST FOR NOMINATIONS TO FILL VACANCIES ON AMA COUNCILS TO THE EXECUTIVE COMMITTEE FOR CONSIDERATION AND ACTION.

Employees Retirement Program

In order to comply with recent ERISA regulations, the following resolution was adopted.

"WHEREAS, The Arizona Medical Association, Inc. adopted an Employee Retirement Plan which was effective February 1, 1969 and executed on February 1, 1969; and,

WHEREAS, the above mentioned Plan is to be replaced by a California-Western States Life Insurance Company Prototype Defined Benefit Plan Document, because of changes required by the Employee Retirement Income Security Act of 1974,

NOW THEREFORE BE IT RESOLVED, that this Company hereby adopt the Plan as proposed to supersede the previous Plan and shall be effective on February 1, 1976; and

RESOLVED FURTHER, that the Trustees of this Plan shall be:

Bruce E. Robinson

and that Bruce E. Robinson shall serve as Plan Administrator of this Plan until further action by this Board; and

RESOLVED FURTHER, that the President of the Company and the above named Trustees be and they hereby are authorized and directed to execute such Retirement Plan Agreement in the name of and on behalf of this Company and do each and every act required in order that this Company may fulfill the terms of the Plan Agreement; and

RESOLVED FURTHER, that the Secretary of this Company be directed to place a copy of such Plan Agreement in the Minutes of this Meeting; and

RESOLVED FURTHER, that at such time as the above mentioned California-Western States Life Insurance Company Prototype has been approved as to form by the National Office of the Internal Revenue Service, and this Company has been so notified by said insurance company, that the President of this Company is hereby directed to apply or cause application to be made to the United States Internal Revenue Service District Office for approval of this Company's superseding Plan and to do everything necessary or proper in order to obtain such approval with the understanding that such amendment shall not affect this Company's previous deductions for contributions already made to said Plan."

It was noted that this resolution in no way alters the content of the retirement plan as adopted by this Board on February 1, 1969 and which is outlined on the following pages.

(*Note: A copy of the plan can be obtained from ArMA offices*).

EXECUTIVE COMMITTEE

Membership Classification

Changes Approved

Maricopa County Medical Society:

(a) Willis J. Nelson, M.D. — Active to Associate — Accounts Retirement — Effective 1/1/78 — Dues Exempt.

(b) C. Thomas Read, M.D. — Active to Associate — Account Retirement — Effective 1/1/78 — Dues Exempt.

(c) Edward A. Smith, M.D. — Active to Associate — Account Residency — Effective 1/1/78 — Dues Exempt.

(d) Maier I. Tuchler, M.D. — Active to Active Over 70 — Account Age — Effective 1/1/78 — Dues Exempt.

(e) Sol Z. Weinzweig, M.D. — Active to Associate — Account Sabbatical — Effective 1/1/78 — Dues Exempt.

Pima County Medical Society:

(a) R. Robert Bates, M.D. — Active to Affiliate — Account Out of State — Effective 1/1/78 — Dues Exempt.

(b) Jorge H. Galindo, M.D. — Active to Affiliate — Account Out of State — Effective 1/1/78 — Dues Exempt.

(c) Otto Gambacorta, M.D. — Active to Associate — Account Retirement — Effective 1/1/78 — Dues Exempt.

(d) Joseph M. Kinkade, M.D. — Active to Active Over 70 — Account Age — Effective 1/1/78 — Dues Exempt.

(e) Oscar S. Koenig, M.D. — Active Over 70 to Associate — Account Retirement — Effective 10/11/77 — Dues Exempt.

Student Memberships Approved

	CLASS
Raymond Andrews	1981
Robert Beren	1979
David Beyer	1979
Robert Cocklin	1979
Keith Comess	1979
Kenneth Davis	1981
Charles Dennis	1979
William Elliot	1981
Bruce Evans	1981
Stephanie Feldman	1981
Kathleen Kolt	1981
Susan Kower	1981
Timothy Schachner	1979
Douglas Shepard	1981
Neal White	1979

FINANCE COMMITTEE

John T. Clymer, M.D., reviewed the financial statements for the period ending October 31, 1977—RECEIVED.

HEALTH CARE COST COMMITTEE, AD HOC

The Board considered the committee's recommendation to seek to have hospitals provide a summary of hospital charges as part of the patient's records so that physicians would have a better understanding of the cost of hospital care.

Many points for and against this approach were expressed.

IT WAS MOVED AND CARRIED TO ENDORSE THE CONCEPT OF INDIVIDUAL HOSPITAL COSTS BEING MADE AVAILABLE TO ALL PHYSICIANS SO THAT THE PHYSICIANS WOULD HAVE A BETTER UNDERSTANDING OF COSTS, BUT THAT THE MECHANISM TO ACCOMPLISH THIS EDUCATIONAL EFFORT BE LEFT TO THE INDIVIDUAL HOSPITAL'S DISCRETION AND INAUGURATION.

IT WAS ALSO MOVED AND CARRIED TO REFER THIS MATTER BACK TO THE COMMITTEE FOR THE PURPOSE OF DRAFTING A RESOLUTION FOR INTRODUCTION IN THE 1978 HOUSE OF DELEGATES THAT WOULD REFLECT THE THOUGHTS SET FORTH IN THE MOTION STATED ABOVE.

LEGISLATIVE COMMITTEE

IT WAS MOVED AND CARRIED TO CONFIRM THE MAIL VOTE TO APPROVE A CAMPAIGN TO GET ALL MEMBERS TO URGE THEIR PATIENTS TO WRITE LETTERS URGING THEIR SENATORS TO VOTE AGAINST POSSIBLE LEGISLATION WHICH WOULD MAKE IT POSSIBLE FOR NON-PHYSICIANS TO DISPENSE AND PRESCRIBE DRUGS. THE VOTE WAS AS FOLLOWS:

19 IN FAVOR
5 AGAINST
2 NOT VOTING

MATERNAL AND CHILD HEALTH CARE COMMITTEE

Birth Defects Registry

IT WAS MOVED AND CARRIED TO SUPPORT THE CONCEPT OF THE ESTABLISHMENT OF A BIRTH DEFECTS REGISTRY IN ARIZONA. THIS SUPPORT DOES NOT INCLUDE FINANCIAL AID AT THIS TIME.

ARIZONA PERINATAL PROGRAM DATA SYSTEM

IT WAS MOVED AND CARRIED TO ENDORSE THE EXTENSION OF THE USE OF THE ARIZONA PERINATAL PROGRAM DATA SYSTEM TO ALL HOSPITALS IN THE STATE AND THAT AN EXTENSIVE PHYSICIAN EDUCATION PROGRAM OF THE DATA SYSTEM BE SUPPORTED.

LEGAL LIABILITY OF MIDWIFERY REGULATIONS

The request that we seek legal counsel opinion on the possible legal liability of physicians as a result of newly proposed Department of Health Services rules relating to the Licensing of Midwifery was discussed at length.

IT WAS MOVED AND CARRIED TO APPROVE THE REQUESTED LEGAL REVIEW, BUT ONLY AFTER OBTAINING AN ESTIMATE OF COST OF SUCH LEGAL REVIEW AND SUBSEQUENT APPROVAL BY THE EXECUTIVE COMMITTEE.

MEDICAL ECONOMICS COMMITTEE

Arizona Physicians Guild

The following resolution submitted to the Board of Directors with the recommendation that the Board introduce it in the 1978 House of Delegates was reviewed in depth.

Subject: THE ECONOMIC ORGANIZATION FOR ARIZONA PHYSICIANS ARIZONA PHYSICIANS GUILD

WHEREAS, The physicians of Arizona need an effective economic organization of bargaining for all levels of physicians who so desire and

WHEREAS, The Arizona Medical Association as constituted can not be an effective economic organization, does not promise to be in the future and can not legally do collective bargaining in its present form and

WHEREAS, Many physicians whether salaried or self-employed want someone to represent them now and

WHEREAS, The self-employed physicians of this state need resolution of economic problems such as; many insurers not performing to contract and Federal and State regulatory bodies determining medical practice economics, therefore be it

RESOLVED: That the Arizona Medical Association in concert with the Professional Guild of Arizona create an independent collective bargaining organization and be it further

RESOLVED: That the presently constituted Medical Economics Committee be extracted from the Arizona Medical Association's standing committees to become the temporary executive committee for the new organization until a general election can be held and be it further

RESOLVED: That the Arizona Medical Association will foster the growth of the new organization by referring economic problems to it (including negotiations with the Industrial Commission of Arizona) and encouraging membership.

IT WAS MOVED AND CARRIED TO NOT HAVE THE ABOVE RESOLUTION INTRODUCED BY THE BOARD, BUT TO REFER IT BACK TO THE COMMITTEE ADVISING THEM THAT THE COMMITTEE COULD INTRODUCE THE RESOLUTION SHOULD IT SO CHOOSE.

PROFESSIONAL COMMITTEE

Statement on Auto Emissions

IT WAS MOVED AND CARRIED TO REAFFIRM THE ASSOCIATION'S POSITION REGARDING AUTOMOBILE EMISSION POLLUTANTS ORIGINALLY ADOPTED October 2, 1976 AS SET FORTH BELOW:

"Every effort must be made to minimize automotive emission pollutants in our air. We must support an effective means of curbing air pollution and creating a safe environment in which we all can live.

There is no question that the health hazards of carbon monoxide and photochemical oxidants can be well demonstrated.

Harmful responses to both of these agents have been found in studies of normal individuals, as well as in patients with chronic diseases. Especially susceptible persons are those with chronic diseases of the heart and lung.

There are large numbers of patients in Arizona with chronic cardiac and respiratory disease in which automotive emission pollutants are clearly hazardous. These same pollutants are potentially dangerous to all persons whatever their state of health.

The Arizona Medical Association strongly supports an effective emissions control program and feels that an effective program to control auto emissions is an important and vital measure for the health of every citizen of Arizona.

Knowing that carbon monoxide and photochemical oxidants are caused from auto emissions, knowing that Arizona is growing and that with it automotive travel will inevitably grow, and knowing that any further increase in pollution levels will be hazardous, every effort must be made to minimize these pollutants.

As a result of these recognized health hazards, the Arizona Medical Association urges the public to continue to support auto emission testing."

ADVANCED CARDIAC LIFE SUPPORT PROGRAM

IT WAS MOVED AND CARRIED TO SUPPORT THE PROFESSIONAL COMMITTEE'S REQUEST TO ENDORSE THE ADVANCED CARDIAC LIFE SUPPORT PROGRAM AS SPONSORED BY THE AMERICAN HEART ASSOCIATION AND TO MAKE THE MEETING FACILITIES OF THE ARIZONA MEDICAL ASSOCIATION AVAILABLE FOR CONDUCTING THE PROGRAMS. THE REQUEST FOR FUNDS ($13,308.00) TO PURCHASE EQUIPMENT FOR THE PROGRAM WAS REFERRED BACK TO THE COMMITTEE TO ATTEMPT TO OBTAIN OTHER SOURCES OF FUNDING.

AMA DELEGATION REPORTS

AMA Convention December 4-7,1977

Written reports were submitted by Victor J. Zannis, M.D., Arthur V. Dudley, Jr., M.D., John F. Kahle, M.D., and Edward Sattenspiel, M.D., copies of which are available to any member requesting same. Verbal reports were also presented by others attending this meeting.

ARIZONA REPUBLIC ARTICLES WRITTEN BY MR. TOM KUHNS

Extensive discussion ensued over the series of articles (11/20/77 - 12/15/77) dealing with various aspects of health care in Arizona.

IT WAS MOVED AND CARRIED TO REFER THIS MATTER TO THE PUBLIC RELATIONS COMMITTEE WITH THE RECOMMENDATION THAT THEY CONSIDER MEETING WITH THE TOP MANAGEMENT OF THE PHOENIX AND TUCSON PAPERS TO SEE IF WE CAN DEVELOP A RELATIONSHIP OF COOPERATION AND ASSISTANCE.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona, on Friday, January 20, 1978, a quorum being present, convened at 6:42 p.m., John F. Kahle, M.D., President and Chairman, presiding.

DEPARTMENT OF HEALTH SERVICES

Dr. Dandoy reported that the Medical Advisory Board for Licensing Drivers had been reactivated with the help of the Association. Much effort and activity is being devoted to solving the problems of the present programs for the retarded. Also, there is a strong effort to find a physician supervisor for the state hospital in cooperation with the Arizona Psychiatric Society. She went on to report that there are efforts being made by the National Health Service Corps to get the Department involved in coordination of the NHSC activities in Arizona.

ARIZONA HEALTH SERVICE CORPS

A review was made of the current status of the AHSC Contract as it relates to HEW's recent retroactive denial to reimburse ArMA for funds paid to Ward B. Studt, M.D.

Mr. Jacobson reviewed his office's comments on the problem along with his recommendations.

IT WAS MOVED AND CARRIED TO EXHAUST ALL AVENUES OF COLLECTING THESE FUNDS FROM HEW BEFORE GOING TO DR. STUDT FOR REIMBURSEMENT.

A TIME FRAME OF THREE MONTHS WAS SET FOR THIS ACTIVITY.

BRITISH NATIONAL HEALTH SERVICE

The committee reviewed the report prepared by Mr. Robinson resulting from his participation in an AMA sponsored week-long seminar on the British National Health Service which was held in London starting November 14, 1977.

The committee determined that the report should get wide distribution.

IT WAS MOVED AND CARRIED TO OBTAIN THE COST OF DISTRIBUTING THE REPORT TO *ALL* ARIZONA PHYSICIANS PLUS SELECTED LEGISLATIVE AND RELATED OPINION MAKERS.

Suggested distribution included:

- Arizona Congressional Delegation
- Health Committees of Arizona Senate & House of Representatives
- Arizona Osteopaths
- Arizona Hospital Association Board of Directors

REQUEST FOR MAILING LIST

Mr. Robinson reported that the Louisiana State Medical Society has requested a mailing list of all Delegates and Alternate

Delegates to the Arizona House of Delegates.

It was pointed out that this was a departure from present policy regarding mailing lists.

IT WAS MOVED AND CARRIED TO REFER THIS MATTER TO THE BOARD OF DIRECTORS FOR ACTION.

PERCENTAGE LEASES

Mr. Jacobson reported on the subject project indicating that additional information would be forthcoming.

MIDWIFERY RULES

Mr. Jacobson reviewed the legal opinion relating to the newly proposed Midwifery Rules being developed by D.H.S. This opinion is being referred to the Maternal and Child Health Care Committee.

U.S. SUPREME COURT DECISION REGARDING ADVERTISING

Mr. Jacobson reviewed the subject matter as follows:

On June 27, 1977, the Supreme Court of the United States decided the case of *Bates et al vs. State Bar of Arizona,* No. 76-316. In its opinion, the Supreme Court recognized advertising by professionals to be a valid exercise of First Amendment rights and, accordingly, not subject to blanket suppression. Nevertheless, the Supreme Court did condone such limited regulation of advertising by professionals as may be necessary to insure accuracy and reliability. Thus, while the Supreme Court approved of advertising which supplied basic informational data, such as name, address, telephone number, office hours and similar routine services and compared the same with charges made by others, it also mentioned the permissibility of regulations which would restrain advertising in a false, deceptive or misleading manner, including advertising the quality of service and advertising conducted by in-person solicitation, as well as advertising procedures and practices which are illegal.

Measured against this standard, the blanket prohibition against advertising found in the Arizona Medical Practice Act is most probably unconstitutional and, consequently, of no force and effect. Therefore, in order to assure that advertising by doctors of medicine flows both freely and cleanly, it is proposed that Arizona Revised Statutes S 32-1401 (10) (c), which presently reads,

"10. 'Unprofessional conduct' shall include the following acts, whether occurring in this state or elsewhere:

(c) Advertising.",

be amended to harmonize with the *Bates* opinion by adding the following emphasized language:

"10. 'Unprofessional conduct' shall include the following acts, whether occurring in this state or elsewhere:

(c) Advertising *in a false, deceptive or misleading manner, advertising the quality of medical service, advertising by in-person solicitation, and advertising procedures and practices which are illegal."*

This language recognizes the right of doctors of medicine to advertise yet sets forth in the exact words of the Supreme Court those advertising practices which are properly subject to restraint.

IT WAS MOVED AND CARRIED TO SEEK INTRODUCTION OF THE PROPOSED AMENDMENT AS DEVELOPED BY OUR SPECIAL COMMITTEE.

AD HOC CLAIM REVIEW COMMITTEE AND AD HOC UNDERWRITING COMMITTEE

IT WAS MOVED AND CARRIED TO INTRODUCE RESOLUTIONS INTO THE HOUSE OF DELEGATES MAKING THESE TWO AD HOC COMMITTEES STANDING COMMITTEES.

BOMEX APPOINTMENTS

Guy B. Atonna, M.D.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE GOVERNOR THE FOLLOWING PHYSICIANS FOR POSSIBLE APPOINTMENT TO THE BOARD OF MEDICAL EXAMINERS TO FILL THE VACANCY CREATED BY THE EXPIRATION OF GUY B. ATONNA, M.D.'s TERM, SUBJECT TO THE PHYSICIAN'S APPROVAL.

M. DAVID BEN-ASHER, M.D.
—TUCSON

ARTHUR V. DUDLEY, JR., M.D.
—TUCSON

JOHN J. STANDIFER, M.D.
—KINGMAN

Albert Eckstein, M.D.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE GOVERNOR THE REAPPOINTMENT OF DR. ECKSTEIN SUBJECT TO HIS AND THE MARICOPA COUNTY MEDICAL SOCIETY'S APPROVAL.

Advisory Committee

IT WAS MOVED AND CARRIED TO EXPLORE THE POTENTIAL OF CREATING AN ADVISORY COMMITTEE TO ASSIST THE GOVERNOR IN MERIT SELECTION OF BOMEX APPOINTEES.

ADVANCED CARDIAC LIFE PROGRAM

Mr. Barnett reported that many people had been contacted seeking funding for this program and as of this date, it appears that we have located funding through the AEMS program.

REQUEST FOR CO-SPONSORSHIP

IT WAS MOVED AND CARRIED TO REJECT THE REQUEST FROM A LOCAL BANK TO CO-SPONSOR A MEDICAL MANAGEMENT PROGRAM.

MEMBERSHIP ON AMA COUNCILS

Discussion ensued on the AMA letter of September 30, 1977 requesting nominations for Councils of the AMA—RECEIVED FOR INFORMATION.

MEMBERSHIP STIMULATION

Mr. Robinson reported on the progress of the subject program indicating that all but three counties had responded. The mailings should occur in February—RECEIVED.

MALPRACTICE PANELS

The request from BOMEX that we supply lists of specialists which would be forwarded to the various county superior court judges for the purpose of selecting malpractice review panel participants was discussed at length.

IT WAS MOVED AND CARRIED TO EMPOWER STAFF TO SEEK SUCH LISTS FROM THE SPECIALTY SOCIETIES AND FORWARD THEM TO BOMEX AS SOON AS POSSIBLE.

CORRESPONDENCE LAWYERS PROFESSIONAL LIABILITY COMMITTEE

A letter of appreciation was received from the State Bar's subject committee thanking us for our offer of assistance to help with the problem of professional liability tort reform—RECEIVED.

OTHER BUSINESS

Voluntary Cost Containment

Mr. Robinson discussed the national effort to get each estate to develop Voluntary Cost Containment programs in cooperation with the hospital associations—RECEIVED FOR INFORMATION.

Newspaper Articles

The subject of how best to assist the newspapers so that the degree of accuracy of articles could be increased was discussed at some length—NO ACTION TAKEN.

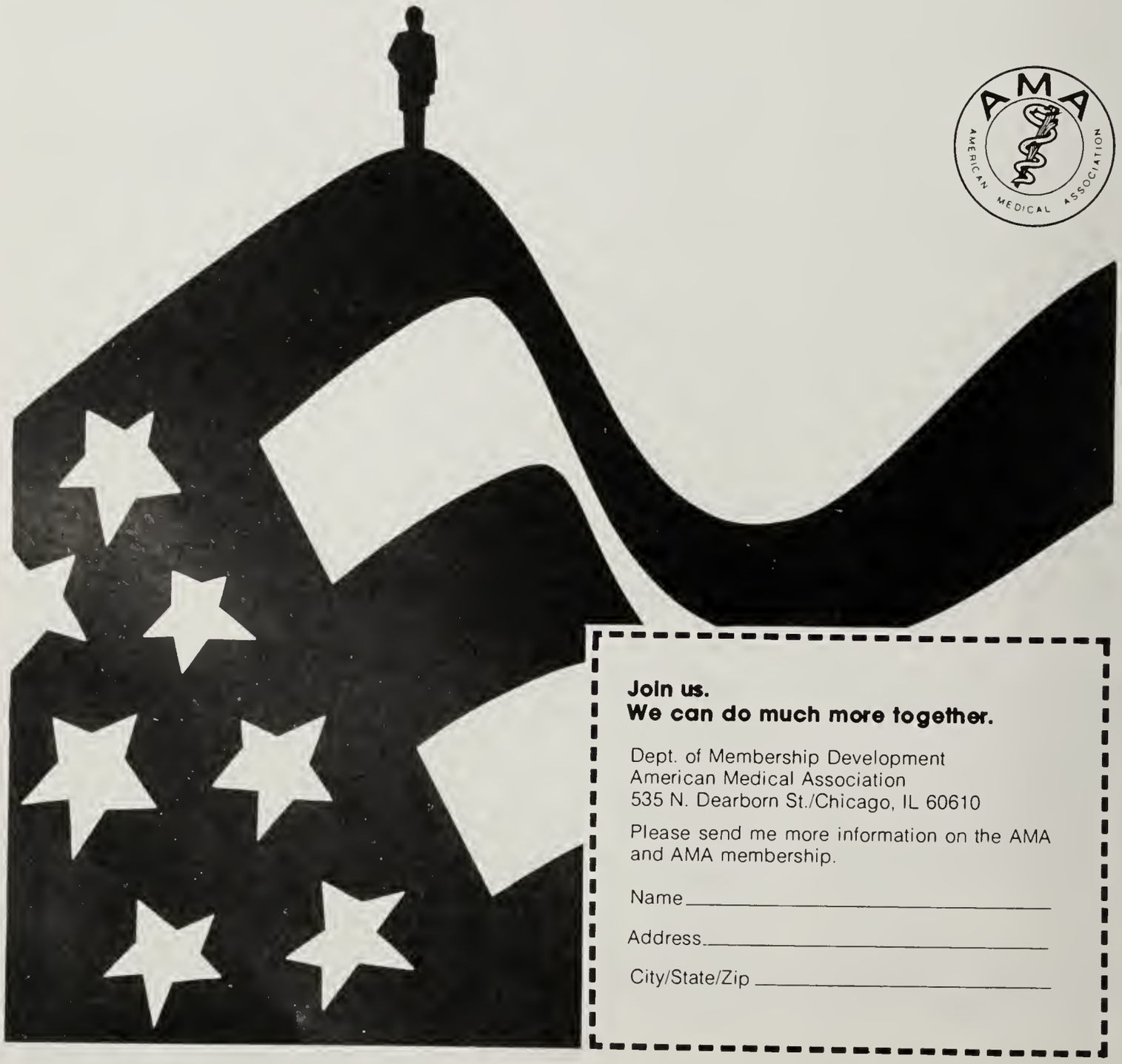
Medicine's men on the Hill

Just who are they? They're the AMA's permanent representatives to the Congress of the United States from the AMA's Washington office.

In every Congress about 10% of all legislation introduced is health related — some 2,500 bills and the number is increasing every year. The AMA lobyists serve as the profession's eyes, ears, and voice on the Hill. Keeping in day-to-day contact with the members of Congress and their staffs. Explaining and promoting the profession's views. Reporting on legislation. And providing legislators with resource material and information on medical and health subjects.

They're on the Hill to protect your interests, lobbying to retain the basic freedoms of medical practice in any government health program that might be enacted. Equally important, they lobby to insure the passage of constructive and workable health legislation for the public.

Sure, the AMA lobbies. It lobbies for the rights and interests of our profession and for quality medical care for every American. With your support, the AMA can be even more effective.



Join us.
We can do much more together.

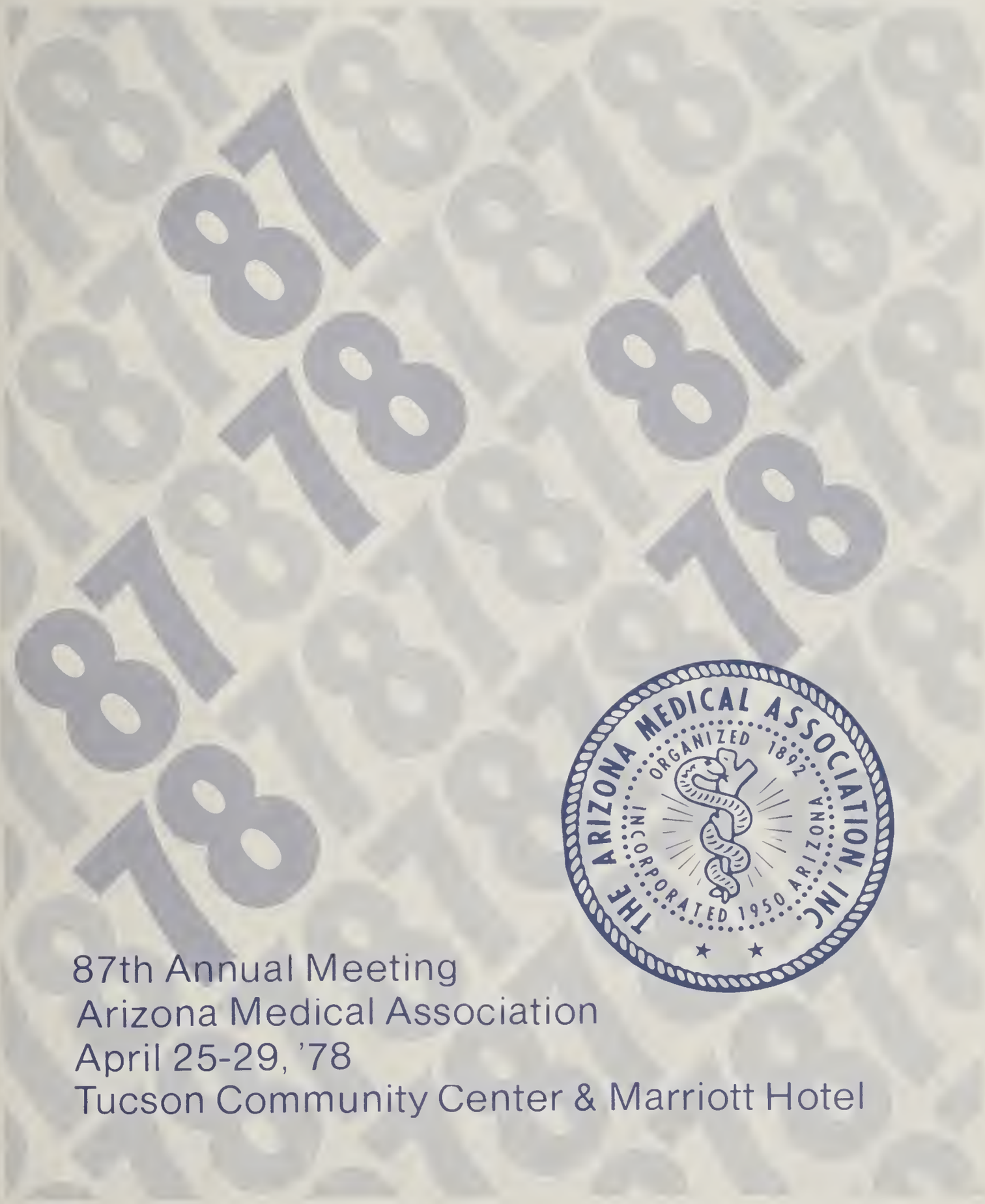
Dept. of Membership Development
American Medical Association
535 N. Dearborn St./Chicago, IL 60610

Please send me more information on the AMA and AMA membership.

Name _____

Address _____

City/State/Zip _____


87th Annual Meeting
Arizona Medical Association
April 25-29, '78
Tucson Community Center & Marriott Hotel

Board of Directors



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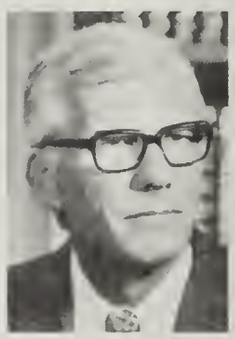
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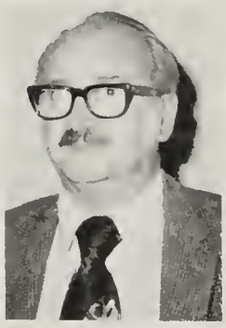
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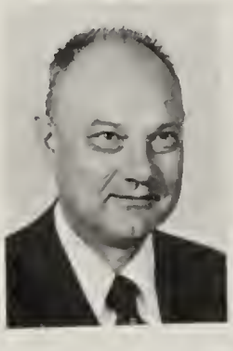
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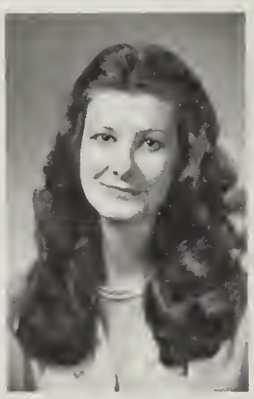
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Secretary to Mr. Robinson



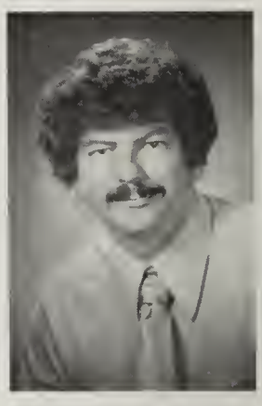
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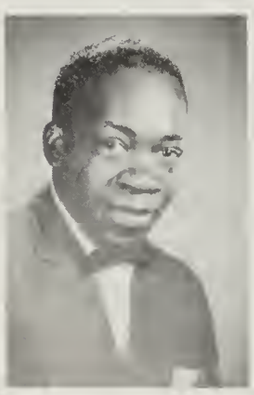
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GOLF TOURNAMENT

Samuel J. Grabb, M.D., Chairman

TENNIS TOURNAMENT

Stuart I. Holtzman, M.D., Chairman

Approved for fourteen and one-half hours required credit for ArMA Certificate in Continuing Medical Education and fourteen and one-half hours elective credit by the American Academy of Family Practice.

Tuesday, April 25, 1978

7:00 AM Executive Committee Breakfast Sonora Room—Marriott

BOARD OF DIRECTORS

9:00 AM Board Meeting Ballroom Salon 3—Marriott
12 Noon Board Luncheon Vista Room—Marriott

HOUSE OF DELEGATES

1:00 PM First General Session Ballroom Salon 1 & 2—Marriott

Call To Order
Robert A. Price, M.D., Speaker

Invocation and Memorial Service
The Reverend Douglas B. Warren
St. Philip's in the Hills, Tucson

Welcome
Robert E. Hastings, Jr., M.D., President
Pima County Medical Society

Introduction of Distinguished Guests
Mrs. Joseph H. Reno, President, Arizona Medical Association Auxiliary
Joseph F. Boyle, M.D., Trustee, American Medical Association

Introduction of Incoming President
John F. Kahle, M.D.

Presidential Address
Richard L. Dexter, M.D.

Regular Business Session of the House
Refer to the AGENDA section of the Delegates Manual

6:30 PM Mutual Insurance Company of Arizona Board of Trustees Dinner Meeting Sonora Room—Marriott

Wednesday, April 26, 1978

8:00 AM ArMA Resolutions Reference Committee Ballroom Salon 3—Marriott

8:00 AM ArMA Bylaws Amendments Reference Committee Durango Room—Marriott

12 Noon to 2:00 PM Specialty Society Luncheons
Arizona Society of Allergists Sonora Room—Marriott

12 Noon Past President's Luncheon Chihuahua Room—Marriott

SPORTS

1:00 PM Annual Golf Tournament
Skyline Country Club
5200 E. St. Andrews Drive, Tucson
$20.00 Fee includes Cart & Green Fees
Registration at the convention required

1:00 PM Annual Tennis Tournament
Tucson Racquet and Swim Club
4001 N. Country Club
Tucson — $3.00 fee
Men's Doubles & Mixed Doubles

7:00 PM Physician Rehabiliation Committee Dinner Meeting Sonora Room—Marriott

6:00 PM Buses Leave for the Tanque Verde Ranch—Lower Lobby Marriott Hotel

6:30 PM Western Steak Fry and Fun at the Beautiful Tanque Verde Ranch in the Rincon Foothills
Dress: Western or casual—Music and great food

10:30 PM Buses Leave for the Marriott Hotel

Thursday, April 27, 1978

7:00 AM Breakfast Ballroom Salon 1—Marriott

7:30 AM to 8:45 AM Breakfast Program for Members and Spouses
Title: The Distressed Physician: Where Do We Go From Here?
Thomas E. Bittker, M.D., Moderator
Stephen C. Scheiber, M.D.
Emanuel M. Steindler

9:00 AM to 12 Noon special event - special event - special event - special event - special event - special event
Computer Assisted Instruction Conquistador Room
Robert C. Combs, M.D. Mezzanine—Marriott
A chance to work out your problems with the assistance of a Boston based computer in these topics: Cardiopulmonary Resuscitation Coma GI Bleeding
Moderator: John I. Hughes, M.D., and faculty from the Emergency Services Department of the College of Medicine, U of A.

Thursday AM	Tucson Community Center Apache Meeting Room (75)	Tucson Community Center Cochise Meeting Room (75)	Tucson Community Center Maricopa Meeting Room (225)	Tucson Community Center Mohave Meeting Room (225)
8 - 9:00 AM				DERMATOLOGY
9 - 9:30 AM	DIABETES AND HYPOGLYCEMIA Grant Gwinup, M.D.	AN ANALYSIS OF DIFFERENTIAL SPINAL ANESTHESIA AND PENTATHOL PAIN STUDY IN THE DIFFERENTIAL DIAGNOSIS OF BACK PAIN John F. Krempen, M.D., Presentor Co-Authors: Richard A. Silver, M.D. James Hadley, Ph.D. Moderator: Burnell R. Brown, M.D.	NEPHROLOGY "Hypertension" Leonard B. Berman, M.D.	Orville J. Stone, M.D. *A six-hour post-graduate course. Advance registration required. Starts at 8 AM and concludes at 5 PM*
9:30 - 10 AM		ROTATIONAL SUBLUXATION OF THE CARPEL NAVICULAR AND INTERCILLARY INSTABILITY OF THE WRIST: A FREQUENTLY MISSED ROENTGEN DIAGNOSIS John C. Bjelland, M.D., Presentor Co-Author: Michael J. Pitt, M.D. Moderator: Gerald Pond, M.D.		
10 - 10:30 AM	Moderator: Rubin Bressler, M.D.	EARLY REHABILITATION AND RETURN TO FUNCTION FOLLOWING RIGID INTERNAL & EXTERNAL FIXATION OF BONE USING THE AO (Swiss) TECHNIQUE Saul N. Schreiber, M.D. Moderator: Donald P. Speer, M.D.		
10:30 - 11 AM	RECESS—Visit Exhibits	RECESS—Visit Exhibits	RECESS—Visit Exhibits	RECESS—Visit Exhibits
11 - 11:30 AM	SPECTRUM OF THYROTOXICOSIS: NEW VARIETIES WHICH ALTER THERAPY Marshall B. Block, M.D., Presentor Co-Authors: Allan I. Cohen, M.D. Laurance B. Nilsen, M.D. John P. Heileman, M.D. Moderator: Charles A. Nugent, M.D.	TREATMENT OF LEUKEMIA—ACUTE AND CHRONIC Lewis M. Slater, M.D. Moderator: Brian G.M. Durie, M.D.	NEPHROLOGY "Evaluation of Urine" Leonard B. Berman, M.D.	DERMATOLOGY Orville J. Stone, M.D. *A six-hour post-graduate course. Advance registration required. Starts at 8 AM and concludes at 5 PM*

Thursday AM	Tucson Community Center Apache Meeting Room (75)	Tucson Community Center Cochise Meeting Room (75)	Tucson Community Center Maricopa Meeting Room (225)	Tucson Community Center Mohave Meeting Room (225)
11:30 - 12 Noon	SPACE AGE DIAGNOSIS OF GALLBLADDER DISEASE M. Herbert Nathan, M.D., Presentor Co-Authors: Marcial M. Ebalo, M.D. Alex Newman, M.D. Moderator: David Earnest, M.D.	TREATMENT OF LEUKEMIA—ACUTE AND CHRONIC Lewis M. Slater, M.D. Moderator: Brian G. M. Durie, M.D.	NEPHROLOGY "Evaluation of Urine" Leonard B. Berman, M.D.	DERMATOLOGY Orville J. Stone, M.D. *A six-hour post-graduate course. Advance registration required. Starts at 8 AM and concludes at 5 PM*

12 Noon to 2:00 PM — Specialty Society Luncheons

Arizona Academy of Family Physicians Sinaloa Room—Marriott
International College of Surgeons Sonora Room—Marriott
Arizona Society of Physical Medicine and Medical Rehabiliation Chihuahua Room—Marriott
Arizona Society of Internal Medicine Durango Room—Marriott
Arizona Urological Society Aztec Room—Marriott

2:00 PM to 5:00 PM — Computer Assisted Instruction Conquistador Room Mezzanine—Marriott
Robert C. Combs, M.D.

A chance to work out your problems with the assistance of a Boston based computer in these topics: Hypertensive Emergencies
Hypertension Management
Hypertension Diagnosis
Fluid and Electrolyte Management

Moderator: John I. Hughes, M.D., and faculty from the Emergency Services Department of the College of Medicine, U of A.

Thursday PM	Tucson Community Center Apache Meeting Room (75)	Tucson Community Center Cochise Meeting Room (75)	Tucson Community Center Maricopa Meeting Room (225)	Tucson Community Center Mohave Meeting Room (225)
2 - 2:30 PM	HYPOXEMIA AS SEEN THROUGH ARTERIAL BLOOD GASES Archie F. Wilson, M.D.	NEONATAL INFECTIONS Gerald R. Greene, M.D.	RADIOLOGY OF THE G.U. TRACT Peter Dure-Smith, M.D.	DERMATOLOGY Orville J. Stone, M.D.
2:30 - 3 PM	Moderator: Benjamin Barrows, M.D.	Moderator: Lawrence J. Fenton, M.D.		*A six-hour post-graduate course. Advance registration required. Starts at 8 AM and concludes at 5 PM*
3 - 3:30 PM	RECESS—Visit Exhibits	RECESS—Visit Exhibits	RECESS—Visit Exhibits	RECESS—Visit Exhibits
3:30 - 4 PM	PATHOPHYSIOLOGY OF ASTHMA Archie F. Wilson, M.D. Moderator: Benjamin Burrows, M.D.	SUSCEPTIBILITY TO MALIGNANT DISEASES: —GENETICS —CHEMICAL MARKERS —RADIATION —DRUG INDUCTION Lewis M. Slater, M.D.	NEPHROLOGY "Urinary Tract Infections and Dialysis Problems" Leonard B. Berman, M.D.	DERMATOLOGY Orville J. Stone, M.D. *A six-hour post-graduate course. Advance registration required. Starts at 8 AM and concludes at 5 PM.*
4 - 4:30 PM	TREATMENT OF ASTHMA Archie F. Wilson, M.D. Moderator: Benjamin Burrows, M.D.			
4:30 - 5 PM	PULMONARY REHABILIATION—WHAT CAN BE DONE Archie F. Wilson, M.D. Moderator: Benjamin Burrows, M.D.	Moderator: David S. Alberts, M.D.		

7:00 PM — ArMPAC Reception Ballroom Salon 2—Marriott
8:00 PM — ArMPAC Annual Banquet Ballroom Salon 1—Marriott

Friday, April 28, 1978

7:00 AM Breakfast Ballroom Salon 1—Marriott

7:30 AM to 8:45 AM Breakfast Program for Members and Spouses
Title: Body Language for Fun and Profit
Donald W. Schafer, M.D.
Assistant Clinical Professor of Psychiatry and Human Behavior
California College of Medicine

9:00 AM to 12 Noon special event - special event - special event - special event - special event - special event
Computer Assisted Instruction Conquistador Room Mezzanine—Marriott
Robert C. Combs, M.D.
A chance to work out your problems with the assistance of a Boston based computer in these topics: Cardiopulmonary Resuscitation Coma GI Bleeding
Moderator: John I. Hughes, M.D., and faculty from the Emergency Services Department of the College of Medicine, U of A.

Friday AM	Tucson Community Center Apache Meeting Room (75)	Tucson Community Center Cochise Meeting Room (75)	Tucson Community Center Maricopa Meeting Room (225)	Tucson Community Center Mohave Meeting Room (225)
8 - 9:00 AM				GASTROENTEROLOGY "A Journey Down the Alimentary Canal" "Upper Gastrointestinal Bleeding" Sheldon S. Zinberg, M.D.
9 - 9:30 AM	RADIOLOGY OF PREGNANCY AND OVARIAN VEIN SYNDROME Peter Dure-Smith, M.D.	CARDIOLOGY *"Floppy" Mitral Valve* Lloyd T. Iseri, M.D.	RENAL TRANSPLANTATION IN CHILDREN: THE PHOENIX EXPERIENCE Karen C. Cartwright, M.D., Presentor Co-Authors: Melvin L. Cohen, M.D. Tracy L. Trotter, M.D. Moderator: Charles F. Zukoski, M.D.	*A six-hour post-graduate course. Advance registration required. Starts at 8 AM and concludes at 5 PM*
9:30 - 10 AM		9:45 AM BASIC PRINCIPLES OF AUSCULTATION	EVALUATION OF MALE INFERTILITY Mark A. Silvert, M.D. Moderator: George W. Drach, M.D.	
10 - 10:30 AM	Moderator: Harlan R. Giles, M.D.	RECESS—10:15 AM 10:30 AM PRESENTATION OF CASES FOR STEHOPHONIC AUSCULTATION Gordon Ewy, M.D. & Lloyd T. Iseri, M.D. 1. "Floppy" Mitral Valve 2. Mitral Stenosis 3. Aortic Stenosis	CURRENT STATUS OF DIAGNOSIS AND TREATMENT OF CARCINOMA OF THE PROSTATE Thomas H. Stanisic, M.D. Moderator: George W. Drach, M.D.	"Dysphagia" Edward R. Dana, M.D.
10:30 - 11 AM	RECESS—Visit Exhibits		RECESS—Visit Exhibits	RECESS—Visit Exhibits
11 - 11:30 AM	THYROID Grant Gwinup, M.D.		TUBERCULOSIS RECOGNITION AND MANAGEMENT Joseph A. Hawkins, M.D. Moderator: Robert A. Barbee, M.D.	NON-MEDICAL ASPECTS OF X-RAY Edward R. Dana, M.D.
11:30 - 12 Noon	Moderator: Thomas W. Boyden, M.D.	Moderator: Gordon Ewy, M.D.	MEDICAL CARE IN THE RURAL COMMUNITIES IN THE STATE STATE OF ARIZONA Dermont W. Melick, M.D. Moderator: Melvin Goodwin, Ph.D.	*An interesting interlude in the post-graduate course on GASTROENTEROLOGY which is a six-hour course starting at 8 AM and concluding at 5 PM*

12 Noon to 2:00 PM Specialty Society Luncheons
Arizona Chapter, American College of Surgeons Sonora Room—Marriott
Arizona Chapter, American Academy of Pediatrics Chihuahua Room—Marriott
Arizona Obstetrical & Gynecological Society Sinaloa Room—Marriott
Arizona Psychiatric Society Vista Room—Marriott
Arizona Thoracic Society Barcelona Room—Marriott

2:00 PM Maternal Mortality Committee Meeting Aztec Room—Marriott

2:00 PM to 5:00 PM Computer Assisted Instruction Conquistador Room Mezzanine—Marriott
Robert C. Combs, M.D.
A chance to work out your problems with the assistance of a Boston based computer in these topics: Hypertensive Emergencies
Hypertension Management
Hypertension Diagnosis
Fluid and Electrolyte Management
Moderator: John I. Hughes, M.D., and faculty from the Emergency Services Department of the College of Medicine, U of A.

Friday PM	Tucson Community Center Apache Meeting Room (75)	Tucson Community Center Cochise Meeting Room (75)	Tucson Community Center Maricopa Meeting Room (225)	Tucson Community Center Mohave Meeting Room (225)
2 - 2:30 PM	OBESITY Grant Gwinup, M.D.		PSYCHIATRY FOR THE NON-PSYCHIATRIST PHYSICIAN Donald W. Schafer, M.D.	GASTROENTEROLOGY "Inflammatory Bowel Disease" Sheldon S. Zinberg, M.D. *A six-hour post-graduate course. Advance registration required. Starts at 8 AM and concludes at 5 PM*
2:30 - 3 PM		PERIOPERATIVE MYOCARDIAL INFARCTION IN AORTOCORONARY BYPASS SURGERY Charles M. Rucker, M.D., Presentor Co-Authors: John Dugall, M.D. Lawrence Ganter, M.D. Mark G. Kartub, M.D. Moderator: Jack G. Copeland, M.D.	Moderator: Alan J. Levenson, M.D.	
3 - 3:30 PM	RECESS—Visit Exhibits	RECESS—Visit Exhibits	RECESS—Visit Exhibits	RECESS—Visit Exhibits
3:30 - 4 PM	REHABILITATION MANAGEMENT OF THE PERSON WITH STROKE Stuart I. Holtzman, M.D. Moderator: William A. Sibley, M.D.	INTESTINAL PARASITES Gerald R. Greene, M.D.	PSYCHIATRY FOR THE NON-PSYCHIATRIST PHYSICIAN Donald W. Schafer, M.D.	GASTROENTEROLOGY "Diverticulitis" Sheldon S. Zinberg, M.D. *A six-hour post-graduate course. Advance registration required. Starts at 8 AM and concludes at 5 PM*
4 - 4:30 PM	INITIAL MANAGEMENT OF HEAD INJURIES Philip R. Weinstein, M.D. Moderator: John I. Hughes, M.D.	Moderator: Kenneth J. Ryan, M.D.		"The Food—Berium Meal" Edward R. Dana, M.D.
4:30 - 5 PM	MULTIPLE SCLEROSIS IN ARIZONA: A CLINICAL PROFILE Colin R. Bamford, M.D. Moderator: William A. Sibley, M.D.	IDENTIFYING THE CAUSATIVE ORGANISM BEFORE THE CULTURE RETURNS Gerald R. Greene, M.D. Moderator: Kenneth J. Ryan, M.D.	Moderator: Alan J. Levenson, M.D.	Questions & Answers Sheldon S. Zinberg, M.D. & Edward R. Dana, M.D.

7:00 PM President's Reception Guadalajara Ballroom—Marriott

8:00 PM President's Banquet Guadalajara Ballroom—Marriott
Guest Speaker: Jokichi Takamine, M.D.
AMA Delegate, California
Dancing to the beautiful music of Ray Starling and his Sounds of Music

Saturday, April 29, 1978

6:00 AM Pima County Medical Society Caucus Breakfast Vista Room—Marriott

HOUSE OF DELEGATES

8:00 AM Second General Session Ballroom Salon 1 & 2—Marriott

12 Noon Board of Directors Meeting and Luncheon Sonora Room—Marriott

Guest Speakers

COLIN R. BAMFORD, M.D.
Tucson

LEONARD B. BERMAN, M.D.
Clinical Professor of Medicine (Nephrology)

THOMAS E. BITTKER, M.D.
Phoenix

JOHN C. BJELLAND, M.D.
Tucson

MARSHALL B. BLOCK, M.D.
Phoenix

KAREN C. CARTWRIGHT, M.D.
Phoenix

ROBERT C. COMBS, M.D.
Associate Dean
Continuing Medical Education
California College of Medicine

EDWARD R. DANA, M.D.
Associate Adjunct Professor of
Radiological Sciences and Medicine
California College of Medicine

PETER DURE-SMITH, M.D.
Professor of Radiological Sciences
California College of Medicine

GERALD R. GREENE, M.D.
Assistant Adjunct Professor of Pediatrics
California College of Medicine

GRANT GWINUP, M.D.
Professor of Medicine (Endocrinology)
California College of Medicine

JOSEPH A. HAWKINS, M.D.
Phoenix

STUART I. HOLTZMAN, M.D.
Tucson

LLOYD T. ISERI, M.D.
Professor of Medicine (Cardiology)
California College of Medicine

JOHN F. KREMPEN, M.D.
Tucson

DERMONT W. MELICK, M.D.
Phoenix

M. HERBERT NATHAN, M.D.
Scottsdale

CHARLES M. RUCKER, M.D.
Phoenix

DONALD W. SCHAFER, M.D.
Assistant Clinical Professor of
Psychiatry and Human Behavior
California College of Medicine

STEPHEN C. SCHEIBER, M.D.
Tucson

SAUL N. SCHREIBER, M.D.
Phoenix

MARK A. SILVERT, M.D.
Tucson

LEWIS M. SLATER, M.D.
Assistant Professor of Medicine
California College of Medicine

THOMAS H. STANISIC, M.D.
Tucson

EMANUEL M. STEINDLER
Director, Department of Mental Health
American Medical Association

ORVILLE J. STONE, M.D.
Professor of Medicine (Dermatology)
California College of Medicine

PHILIP R. WEINSTEIN, M.D.
Tucson

ARCHIE F. WILSON, M.D.
Associate Professor of Medicine (Pulmonary)
and Chief of Pulmonary Diseases
California College of Medicine

SHELDON S. ZINBERG, M.D.
Assistant Clinical Professor of Medicine
(Gastroenterology)
California College of Medicine

Presidents



Mrs. Joseph H. Reno
1977-78



Mrs. James L. Parsons
1978-79

Convention Co-Chairwomen

Mrs. Lawrence Adler

Mrs. Thomas E. Newman

Highlights Desert Pow Wow

TUESDAY, APRIL 25, 1978

9 AM - 4 PM — Registration, Hospitality, AMA-ERF Exhibit Hall—Lower Lobby—Marriott

11 AM - 2 PM — Arts & Crafts Show set up

1:00 PM — ArMA House of Delegates Ballroom 1 & 2—Marriott
ArMA Auxiliary President's Address
Mrs. Joseph H. Reno
American Medical Association Trustee
Joseph F. Boyle, M.D.

4:00 PM — Judging of Arts & Crafts Show

WEDNESDAY, APRIL 26, 1978

9 AM - 4 PM	Registration, Hospitality, AMA-ERF Exhibit Hall—Lower Lobby—Marriott Arts & Crafts Show Marriott
9:30 AM	Health Education Committee & County Presidents (1978-79) Aztec Room—Marriott
11:00 AM to 5:00 PM	Trip to Nogales, Mexico Lower Lobby—Marriott
6:00 PM	Buses Leave for the Tanque Verde Ranch—Lower Lobby Marriott Hotel
6:30 PM	Western Steak Fry and Fun at the Beautiful Tanque Verde Ranch in the Rincon Foothills *Dress: Western or casual—Music and great food*
10:30 PM	Buses Leave for the Marriott Hotel

THURSDAY, APRIL 27, 1978

7 - 8:45 AM	Breakfast Program for ArMA Members and Spouses Ballroom Salon 1—Marriott Title: The Distressed Physician:Where Do We Go From Here? Thomas E. Bittker, M.D., Moderator Stephen C. Scheiber, M.D. Emanuel M. Steindler
8:00 AM	Pre-Convention Board Meeting Durango Room—Marriott
9 AM - 4 PM	Registration, Hospitality, AMA-ERF Exhibit Hall—Lower Lobby—Marriott Arts & Crafts Show Marriott
9:00 AM	Complimentary Breakfast for Ballroom Salon 2—Marriott incoming and out-going state officers, chairwomen, and county presidents. Purpose: Exchange guide books and materials.
10 AM - 12 Noon	First General Session Ballroom Salon 3—Marriott All Auxiliary Members Invited Election of Officers to be followed by Executive Committee Meeting (1978-79) to be announced Nominating Committee Meeting (1978-79) to be announced
10 AM - 12 Noon	Morning Walking Tour of Downtown Historic Tucson—Guides will be provided Upper Lobby—Marriott
12:30 PM	Gavel Club Luncheon (Past-Presidents) Barcelona Room—Marriott
1 - 3 PM	Afternoon Walking Tour of Downtown Historic Tucson—Guides will be provided Upper Lobby—Marriott
2:30 PM	Hamer Fund Committee Aztec Room—Marriott
7 PM	ArMPAC Reception Ballroom Salon 2—Marriott
8 PM	ArMPAC Banquet Ballroom Salon 1—Marriott

FRIDAY, APRIL 28, 1978

7 - 8:45 AM	Breakfast Program for ArMA Members and Spouses Ballroom Salon 1—Marriott Title: Body Language for Fun and Profit Donald W. Schafer, M.D. Assistant Clinical Professor of Psychiatry and Human Behavior California College of Medicine
8:30 - 11 AM	Registration, Hospitality, AMA-ERF Exhibit Hall—Lower Lobby—Marriott
8:30 AM - 12:30 PM	Arts & Crafts Show Marriott

9 - 11 AM	Second General Session Ballroom Salon 3—Marriott All Auxiliary Members Invited Memorial Service AMA Auxiliary President's Report Mrs. Chester L. Young ArMA Auxiliary President's Report Mrs. Joseph Reno
11:30 AM - 12:30 PM	Reception (no host cocktails) Ballroom Salon 2—Marriott Viewing Arts & Crafts Show
12:30 PM	Installation Luncheon & Fashion Show Ballroom Salon 2—Marriott ArMA President's Address Richard L. Dexter, M.D. Greeting from AMA Auxiliary President Mrs. Chester L. Young Installation of Officers Acceptance Speech—Mrs. James L. Parsons to be followed by Post Convention meeting of New Board of Directors Durango Room—Marriott
2:30 - 3:30 PM	Pick Up Arts & Crafts Exhibits
7:00 PM	ArMA President's Reception Guadalajara Ballroom—Marriott
8:00 PM	ArMA President's Banquet Guadalajara Ballroom—Marriott Guest Speaker: Jokichi Takamine, M.D. AMA Delegate, California *Dancing to the beautiful music of Ray Starling and his Sounds of Music*

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

THE FOLLOWING INSTITUTIONS AND ORGANIZATIONS HAVE RECEIVED ACCREDITATION FOR CONTINUING MEDICAL EDUCATION FROM THE COUNCIL ON MEDICAL EDUCATION OF THE AMA

ARIZONA MEDICAL ASSOC., INC.
ARIZONA STATE HOSPITAL, PHOENIX
DESERT SAMARITAN HOSPITAL, MESA
GOOD SAMARITAN HOSPITAL, PHOENIX
HEALTH MAINTENANCE ASSOCIATES
PHOENIX INDIAN MEDICAL CENTER
PHOENIX PSYCHIATRIC COUNCIL
MARICOPA COUNTY GENERAL HOSPITAL, PHOENIX
MEMORIAL HOSPITAL, PHOENIX
ST. LUKE'S HOSPITAL AND MEDICAL CENTER, PHOENIX
ST. JOSEPH'S HOSPITAL AND MEDICAL CENTER, PHOENIX
TUCSON HOSPITALS MEDICAL EDUCATION PROGRAM, TUCSON
U OF A HEALTH SCIENCES CENTER
VETERANS ADMINISTRATION CENTER, PRESCOTT
VETERANS ADMINISTRATION HOSPITAL, PHOENIX

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category I credit. Physicians who participate in programs which are designated Category I by accredited institutions will receive Category I credit toward the ArMA Certificate in CME and the AMA's Physicians Recognition Award.

MARCH

2ND ANNUAL SEMINAR, CURRENT CONCEPTS OF HYPERTENSION.
March 11-12, 1978. Scottsdale Registry Resort, Scottsdale, AZ. Sponsor: Health Maintenance Associates. Contact: Dr. L. A. Oslen, Health Maintenance Associates, 4747 N. 22nd St., Phoenix, AZ 85016. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

CURRENT CONCEPTS IN CEREBRO-VASCULAR DISEASE.
March 11-18, 1978. Sun Valley Inn, Sun Valley, Idaho. Contact: Margo Walter, Administrative Assistant, Office of Continuing Medical Education and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 22.5 required hours toward the ArMA Certificate in Continuing Medical Education.

GASTROENTEROLOGY FOR CLINICIANS — 1978
March 13-15, 1978. Del Webb's Towne-House, Phoenix, AZ. Sponsor: American College of Gastroenterology & the Institute of Gastroenterology, Good Samaritan Hospital. Contact: David C. H. Sun, M.D., Good Samaritan Hospital, Phoenix, AZ 85036. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL EDUCATION LECTURE
April 11, 1978, 7 a.m., conference room, Glendale Samaritan Hospital. Sponsor: Glendale Samaritan Hospital. Contact: Richard Haynes, M.D., GSH, 6010 W. Northern, Glendale, AZ 85301. Approved for one required hour toward the ArMA Certificate in Continuing Medical Education.

"MANDATORY REPORTING OF CHILD ABUSE"
March 18, 1978, Arizona Medical Association offices, Phoenix. Sponsor: Psychiatric Services, Ltd. Contact: Robert T. Dean, Jr., M.D., Ste. 121, 2202 E. Camelback Rd., Phoenix, AZ 85016.

MEDICAL HISTORY SOCIETY OF ARIZONA
Wednesday, March 20, 1978, 7:30 p.m. Contact William Weese, M.D., President, 550 W. Thomas Road, Phoenix, AZ 85013.

CLINICAL RECOGNITION AND MANAGEMENT OF HEART DISEASE 1978
March 23-25, 1978. Auditorium, AHSC, Tucson, AZ. Sponsor: Dept of Cardiology, and Internal Medicine, U of A Health Sciences Center. Contact: Frank I. Marcus, M.D., 1501 N. Campbell, Tucson, AZ. Approved for 20 required hours toward the ArMA Certificate in Continuing Medical Education.

14th ANNUAL ARIZONA CHEST SYMPOSIUM
March 31, April 1, 2, 1978. Doubletree Inn, Tucson, AZ. Sponsor: U of A Health Sciences Center, Tucson Medical Center. Contact: Charles E. Blair, M.D., Carl F. Diener, M.D., Tucson Veterans Administration Hospital, Tucson, AZ. Approved for 20 required hours toward the ArMA Certificate in Continuing Medical Education.

APRIL

COMBINED GROUND ROUNDS
March 1, 1978, St. Joseph's Hospital. Sponsor: Family Practice Residency Programs of St. Joseph's, Good Samaritan, and Scottsdale Memorial Hospitals. Contact: Ann Larsen, R.N., Program Coordinator, Family Practice Resic'ency Program, St. Joseph's Hospital and Medical Center, Phoenix, AZ 85013. Approved for required hours toward the AMA Certificate in Continuing Medical Education.

ISSUES IN CRITICAL CARE MEDICINE
April 8, 1977. Arizona State University. Sponsor: Desert Samaritan Hospital, Contact: L. A. Rosati, M.D., Dept. of Path., Desert Samaritan Hospital. Approved for 7 required hours toward the ArMA Certificate in Continuing Medical Education.

GERIATRIC POW-WOW
April 7, 8, 9, 1978. Arizona Inn, Tucson, AZ. Sponsor: Continuing Medical Education, U of A. Contact: A. Vuturo, M.D. Office of Continuing Medical Education and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 16 required hours toward the ArMA Certificate in Continuing MedicEl Education.

MEDICAL EDUCATION LECTURE
March 14, 1978, 7 a.m., Conference Room, Glendale Samaritan Hospital. Sponsor: Glendale Samaritan Hospital. Contact: Richard Haynes, M.D., 6010 W. Northern, Glendale, AZ 85301. Approved for one required hour toward the ArMA Certificate in Continuing Medical Education.

PEDIATRIC RADIOLOGY FOR THE GENERAL RADIOLOGIST
April 14-16, 1978, Doubletree Inn at Scottsdale Mall, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contact: A.R. Sandrock, M.D., Chairman, Dept. of Radiology, Maricopa County General Hospital, 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 15 required hours toward the ArMA Certificate in Continuing Medical Education.

ARIZONA MEDICAL ASSOCIATION 86th ANNUAL CONVENTION.
April 25-29, 1978, Tucson Community Center and Marriott Hotel, Tucson, AZ. Sponsor: Arizona Medical Association, Inc. Contact: Mr. Bruce E. Robinson, 810 W. Bethany Home Rd. Phoenix, AZ 85013. Approved for required hours toward the ArMA Certificate in Continuing Medical Education. Detailed program will appear in the March issue of *Arizona Medicine.*

AMBULATORY PEDIATRIC ASSOCIATION, INC. 18TH ANNUAL MEETING
April 24, 25, 1978. New York Hilton, N.Y., N.Y. Sponsor: Ambulatory Pediatric Association, Inc. Contact: Roberta Weiss, 4525 E. San Francisco St., Tucson, AZ 85210. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

ARIZONA UROLOGICAL SOCIETY LUNCHEON
April 27, 1978, 12 Noon, Marriott Hotel, Tucson, AZ. Sponsor: Arizona Urological Society. Contact: Everett D. Hendricks, M.D., 1055 Ruth Street, Prescott, AZ 86301. Murray Feldstein, M.D., 900 N. San Francisco St., Flagstaff, AZ 86001.

MAY

UPDATE: PRIMARY CARE 1978
May 3-6, 1978. Arizona Health Sciences Center Sponsor: U of A Continuing Medical Education. Contact: A. Vuturo, M.D., Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 25 required hours toward the ArMA Certificate in Continuing Medical Education.

RURAL HEALTH/EMERGENCY CARE CONFERENCE
May 12, 13, 1978. Little America, Flagstaff, AZ. Sponsor: U of A Continuing

Medical Education Program. Contact: John Hughes, M.D., Emerg. Serv. Dept., or Anthony Vuturo, M.D., Continuing Medical Education and Outreach, U of A Health Sciences Center. Tucson, AZ 85724. Approved for 12 required hours toward the ArMA Certificate in Continuing Medical Education.

SYMPOSIUM ON RADIATION PATHOLOGY
May 22, 23, 24, 1978. Wesward Look Resort, Tucson, AZ. Sponsor: U of A Health Sciences Center. Contact: David White, M.D., Anthony F. Vuturo, M.D., UAHSC, 1501 N. Campbell Ave., Tucson, AZ. Approved for 18 required hours toward the ArMA Certificate in Continuing Medical Education.

JUNE

17th ANNUAL STATE CONVENTION
Maricopa Chapter, American Association of Medical Assistants, June 9, 10, 11, 1978, Doubletree Inn, Scottsdale, AZ. Sponsor: Maricopa Chapter, American Association of Medical Assistants. Contact: Jan Neal, CMA Convention Chairman, 4502 N. 36th St., $108, Phoenix, AZ 85018.

MEDICAL HORIZONS: ADVANCES IN THERAPEUTICS
June 30-July 2, 1978. Little America Hotel, Flagstaff, AZ. Sponsor: Office of Continuing Medical Education, U of A Health Sciences Center, Tucson, AZ. Contact: Anthony F. Vuturo, M.D., AHSC, Tucson, AZ 85724. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

JULY

21st ANNUAL RUIDOSO FAMILY PRACTICE SEMINAR
July 17-20, 1978. Headquarters Inn, of the Mountain Gods at Mescalero, New Mexico. Sponsors: New Mexico Chapter of the American Academy of Family Physicians. Contact: Bob Reid, Convention Dir., P. O. Box 456, Sunland Park, N.M. 88063. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

FIRST ANNYAL SUMMER PEDIATRIC CONFERENCE PREPARATION FOR RECERTIFICATION
July 7, 8, 9, 1978. 7:30 a.m.-2 p.m., Little America, Flagstaff, AZ. Sponsor: CME, U of A Health Sciences Center. Contact: Anthony F. Vuturo, M.D., ASHSC, 1501 N. Campbell Ave., Tucson, AZ 85724. Approved for 17.5 required hours toward the ArMA Certificate in Continuing Medical Education.

MONTHLY OR WEEKLY

HEADACHE ROUNDS
Tuesday afternoons or Thursday mornings weekly. Sponsor: Headache Group of the Southwest. Contact: Peggy Voigt, 2610 W. Bethany Home Rd., Phoenix, AZ 85017, or phone 242-6646.

SHRINE MEDICS MEETING
Second Tuesday of each month, Doctors Hospital, 5:45 p.m., J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

OFFICE PSYCHIATRY FOR THE PRIMARY PROVIDER
2nd Monday of month, 4811 N 7th Street, Phoenix, AZ Sponsor Arizona Health Plan Contact T R Bittker, M D , Box 5000, Phoenix, AZ 85010 Approved for required hours toward the ArMA Certificate in Continuing Medical

COUNTER TRANSFERENCE GROUP
Weekly, Thurs 8-10 p m Sponsor Phoenix Psychiatric Council Contact James E Campbell, M D., 5051 N 34th St., Phoenix, AZ 85018 Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education.

MONTLHY MEETING OF TUCSON RADIOLOGISTS
Last Tues of Month, Plaza International, Tucson, AZ Sponsor U of A Medical Center, Dept. of Radiology Contact Irwin M. Freundlich, M D., Arizona Medical Center, Dept. of Radiology, Tucson, AZ 85724 Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education

PEDIATRIC GRAND ROUNDS
Tuesday 7:30 - 8:30 a.m. in Phoenix: 1st Tue. - Phoenix Indian Hospital, 2nd Tue. - Maricopa County Hospital, 3rd Tue. - Good Samaritan Hospital, 4th Tue. - St. Joseph's Hospital
Sponsor: Phoenix Hospital's Affiliated Pediatric Program. Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

REVIEW OF FORENSIC PATHOLOGY, CURRENT CASE, SPECIAL TOPICS
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists, Contact: H.H. Karnit Schinig, M.D., 120 S. 6th Ave. Phoenix, AZ. Approved for 1 required hour per session toward ArMA CME Certificate.

Arizona State Hospital

STAFF EDUCATION CONFERENCE
Wednesdays. Weekly, 1 p m Arizona State Hospital, Phoenix, AZ Sponsor Arizona State Hospital Contact Howard E. Wulsin, M D , Arizona State Hospital, 2500 E Van Buren, Phoenix, AZ 85008 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

Baptist Hospital

SURGICAL GRAND ROUNDS
4TH TUESDAY OF EACH MONTH
Hospital Auditorium, Baptist Hospital, Phoenix. Sponsor Baptist Hospital Phoenix Contact James B Shields, M D , 6036 N 19th Ave., Phoenix, AZ 85015 Approved for 1-1/2 required hours per month toward the ArMA Certificate in Continuing Medical Education.

Barrow Neurological Institute

MEDICAL EDUCATION
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center. 350 W. Thomas Road, phoenix, AZ 85013. Contact: John R. Green, M.D., Director. Approved for one hour required credit toward the ArMA Certificate in Continuing Medical Education.

NEUROLOGY TEACHING CONFERENCE
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

NEUROSURGERY MORBIDITY CONFERENCE
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.

SPINAL INJURY CONFERENCE
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Room—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists.

NEUROPATHOLOGY OF GROSS SPECIMENS CONFERENCE
Thursdays, 7:30-8:30 a.m. in the Morgue.

NEUROLOGY-NEUROSURGICAL CLINICAL-NEURORADIOLOGICAL AND NEUROPATHOLOGICAL CONFERENCE
Fridays, 8-9 a.m., First Floor Conference Room.

NEUROPATHOLOGY OR NEURORADIOLOGY CONFERENCES
Fridays, 9 a.m., Neuropathology in Neuropathology Conference Room, Neuroradiology in First Floor Conference Room.

NEUROSURGICAL JOURNAL CLUB
Saturdays, 9-11 a.m., in Eighth Floor Conference Room.

Camelback Hospital

PATIENT STAFFING CONFERENCE
Three times weekly. Camelback Hospital Phoenix, AZ. Sponsor Camelback Hospital Contact Medical Director, Camelback Hospital, 5055 N. 34th St., Phoenix, AZ 85018 Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

Good Samaritan Hospital

DESERT SAMARITAN HOSPITAL
Wednesday Evenings 7 p m Sponsor Desert Samaritan Hospital Contact L A Rosati, M D . Laboratory Desert Samaritan Hospital, Mesa. AZ 85202 Approved for required hours toward the ArMA Certificate in Continuing Medical Education

CLINICAL CANCER CONFERENCE
3rd Wednesday every month, Butler Bldg Conference Room Good Samaritan Hospital, Phoenix, AZ Sponsor Good Samaritan Hospital Contact John A Bruner M D 926 E McDowell Road, Phoenix, AZ 85006 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

PULMONARY DISEASE GRAND ROUNDS
Mondays—12 Noon. D-5 North Conference Rm . Good Samaritan Hospital, Phoenix, AZ Sponsor Pulmonary Disease Teaching Service, Good Samaritan Hospital Contact Bernard E Levine, M D . Pulmonary Function Laboratory, Good Samaritan Hospital, 1033 E McDowell Hospital, Phoenix, AZ 85006 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

CONTINUING MEDICAL EDUCATION PROGRAM
Maryvale Samaritan Hospital, every other Wednesday, 7-8 a.m., January 25, 1978 through December 27, 1978. Sponsor: Medical Staff of Maryvale Hospital. Contact: Richard C. Christensen, M.D. Approved for one required hour per session toward the ArMA Certificate in Continuing Medical Education.

Memorial Hospital, Phoenix

MONTHLY MEDICAL EDUCATION SEMINAR
Third Monday of the Month, 6 30 p m . Kiva Conference Room, Phoenix Memorial Hospital Sponsor Medical Staff of Memorial Hospital Contact George Scharf, M D , 1201 South 7th Avenue, Phoenix, AZ 85007 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

TUMOR BOARD CONFERENCE
Every Friday at Noon, Kiva Conference Room, Phoenix Memorial Hospital Sponsor Phoenix Memorial Hospital Contact Howard Kimball, M D , 333 West Thomas Road, Phoenix, AZ 85013 Approved for credit toward the ArMA Certificate in Continuing Medical Education

Scottsdale Memorial Hospital

CLINICAL PATHOLOGICAL CONFERENCE
4th Monday, Monthly, 12 45 p m , Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor Medical Staff Contact R C Good, M D , Director of Medical Education, 7300 E 4th St , Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

FAMILY PRACTICE CONFERENCE
1st Monday Monthly, 12 45 p m , Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor Medical Staff Contact R C Good, M D , Dir of Medical Education, 7300 E 4th St , Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

MEDICAL GRAND ROUNDS
3rd Monday, Monthly, 12 45 p m , Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor Medical Staff Contact R C Good M D , Dir of Medical Education, 7300 E 4th St , Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

MORBIDITY & MORALITY CONFERENCE
2nd Monday, Monthly, 12 45 p m , Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor Medical Staff Contact R C. Good, M D , Dir Medical Education, 7300 E 4th St , Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education.

St. Mary's Hospital, Tucson

CARDIOLOGY CONFERENCE
Weekly—Friday 8-9 a.m , St Mary's Hospital Auditorium, Tucson, AZ Sponsor St Mary's Hospital Contact A L Forte, M D . St Mary's Hospital, Tucson, AZ 85724 Approved for one required hour toward the ArMA Certificate in Continuing Medical Education.

GRAND ROUNDS
Each Thursday 7 a m -8 a m , St Mary's Hospital and Health Center, Sponsor Depts of Medicine, Surgery, Radiology, Pathology and Family Practice Contact Richard Silver, M D . Chairman, Medical Education and Library Committee, Century Medical Plaza, Suite 160, 1701 West St Mary's Road Tucson, AZ 85703 Approved for 1 required hour per round toward the ArMA Certificate in Continuing Medical Education

MONTHLY SPECIALTY CONFERENCE OF THE DEPARTMENT OF SURGERY
First Monday, each month, 7:30 a.m., Century Room A. Sponsor: St. Mary's Hospital & Health Center. Contact: Medical Staff Office, St. Mary's Hospital & Health Center, P.O. Box 5386, Tucson, AZ 85703. Approved for one required hour per session toward the ArMA Certificate in Continuing Medical Education.

U of A Health Sciences Center

CLINICAL IMMUNOLOGY, ALLERGY AND RHEUMATOLOGY ROUNDS
Every Friday Noon-1 p.m. Sponsor U of A College of Medicine, Dept of Internal Medicine, Clinical Immunology Section Contact John Boyer M D , U of A College of Medicine, Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

CEREBROVASCULAR DISEASE CONFERENCE
Mondays 5-6 p.m., weekly, Rm 5505 Surgery, U of AHSC. Sponsor: Depts. of Surgery, Neurology and Radiology, UAHSC. Contact: Dr. Wesley Moore, U of A Health Sciences Center, Tucson, AZ 85724.

ENDOCRINOLOGY SEMINAR
Every Thursday Noon-1 p m 1st 3rd & 5th Thursday—Rm N318, VA Hospital 2nd & 4th Thursday, Rm 6505, Tucson Medical Center Sponsor U of A College of Medicine, Department of Internal Medicine Department of Internal Medicine, Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

HEMATOLOGY-ONOCOLOGY CLINICAL CONFERENCE
Every Tuesday, Noon-1 p m 1st, 3rd & 5th Tuesdays—Rm 6505 AZ Medical Center 2nd & 4th Tuesdays—Rm N318 Veterans Adm Hospital Sponsor U of A College of Medicine, Dept of Internal Medicine Contact Sidney Salmon M D , U of A College of Medicine, Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

MEDICAL GRAND ROUNDS
Every Wednesday Noon-1 p m 1st, 3rd & 5th Wednesday—Staff Conf Rm VA Hospital 2nd & 4th Wednesday—Rm 5403 Arizona Medical Center Sponsor U of A College of Medicine, Dept of Internal Medicine Contact Jay Smith, M D , U of A College of Medicine, Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

NUCLEAR MEDICINE
Thursday, 7:30 a.m., Radiology Conf. Rm., AHSC, Tucson. Sponsor: Dept. of Diagnostic Radiology. Contact: Irwin M. Freundlich, U of A Health Sciences Center, Tucson, AZ 84724. Approved for one required hour per session toward ArMA CME Certificate.

NEUROLOGY GRAND ROUNDS
Every Friday, 12 noon. 1st, 3rd & 5th Fridays, U of A Health Sciences Center, Rm. 7505. 2nd and 4th Fridays, Veterans Administration Hospital. Sponsor: U of A Health Sciences Center, Dept. of Neurology. Contact: William A. Sibley, M.D., Dept. of Neurology U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

NEUROPATHOLOGY REVIEW CONFERENCE
Thursdays, 8:30 a.m., Rm 5120 Arizona Health Sciences Center. Sponsor: U of A College of Medicine. Contact: Peter C. Johnson, M.D., 1501 N. Campbell Ave., Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

NEUROSCIENCES SEMINAR
Every Friday, 7:30 a.m. U of A Health Sciences Center, Rm. 7505, Tucson, AZ. Sponsor: Dept. of Neurology. Contact: Colin R. Bamford, M.D. & Jose F. Laguna, M.D., Dept. of Neurology, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PATHOLOGY SEMINAR
Friday, 4:30-5:30 p.m., Rm 5120 AHSC. Tucson, AZ. Sponsor: Dept. of Pathology, Contact: Dr. Kenneth Ryan, Dept. of Path. U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour per session toward the ArMA Continuing Medical Education Certificate.

TUCSON PATHOLOGIST CONFERENCE
First Monday of the month, 7:30-9 p.m., Rotation Between Tucson Pathology Conf. Rms., Sponsor: Tucson Pathologists. Contact: Dr. Merle Clard, U of A Health Sciences Center, Dept. of Path., Tucson, AZ 84724. Approved for 1 required hour per session toward the ArMA Certificate in CME.

PEDIATRIC GRAND ROUNDS
Every Tuesday, 12:30 p.m. - 1:30 p.m. Alternates between Tucson Medical Clinic and U of A Health Sciences Center. Sponsor U of A College of Medicine, Dept. of Pediatrics. Contact: Hugh Thompson, M.D., 1501 N. Campbell, Tucson, AZ. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PROBLEM CASE WORKSHOPS
3rd Monday of each month 7 30 a m. Room 4410, Arizona Medical Center, Tucson, AZ Sponsor Division of Ophthalmology. U. of A College of Medicine Contact H E Cross. M.D., Ph.D Arizona Medical Center. Dept of Surgery. Tucson, AZ Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education

PSYCHIATRIC GRAND ROUNDS
Every Wed. Sept to May. 4-5 30 p m Rm 8403, Arizona Medical Center. Tucson, AZ Sponsor U of A College of Medicine Dept of Psychiatry Contact Alan Levenson, M.D. U of A College of Medicine, Tucson, AZ 85721 Approved for 1-1/2 required hour per session toward the ArMA Certificate in Continuing Medical Education

CHEST RADIOLOGY
Monday, 5-6 p.m., weekly, Rm. 1535F, AHSC, Tucson, AZ. Sponsor: Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of Radiology, Tucson, AZ 85724. Approved for 1 required hour per session toward ArME CME Certificate.

GASTROINTESTINAL RADIOLOGY
Mondays, 7:30 a.m., weekly except for summer months. Sponsor: Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., Diagnostic Radiology, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour toward ArMA CME Certificate.

RADIATION ONCOLOGY PLANNING CONFERENCE
Friday, weekly, 8:30-10 a.m., AHSC Rm. 0655. Sponsor: Radiation oncology Section, UAHSC, Tucson, AZ. Contact: Robert S. Heusinkveld, M.D., Ph.D., U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1.5 required hours per session toward ArMA Certificate in CME.

RADIOLOGY-RHEUMATOLOGY CONFERENCE
Thursday, 8 a.m., UAHSC, Library, Rm. 1535C. Sponsor: Dept. of Radiology, Contact: Irwin M. Freundlich, U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour toward the ArMA Certificate in CME.

VASCULAR RADIOLOGY
January thru June 1978. 7:30 a.m. to 8:30 a.m. every Friday. Dept. of Radiology. Sponsor: Dept. of Radiology, U of A Health Sciences Center. Contact: I. M. Freundlich, M.D., U of A Health Sciences Center Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

RADIOLOGY INTERESTING CASE CONFERENCE
Thursday, noon, Veterans Administration Hospital, Tucson, AZ. Sponsor: U of A Health Sciences Center, Tucson, Radiology Dept. Contact: Irwin M. Freundlich, M.D., U of A health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA CME Certificate.

RESIDENT'S CONFERENCE
Wednesday/weekly 5-6 p.m., Diag. Radiology Conf. Rm., U of A Health Sciences Center. Sponsor: U of A Health Sciences Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour per session toward the ArMA Certificate in Continuing Medical Education.

TRAUMA CONFERENCE
Every Thursday, 4-5:00 p.m. Health Sciences Center, Rm. 5505, Tucson, AZ. Sponsor: U of A College of Medicine, Dept. of Surgery. Contact: Martin Silverstein, M.D., Dept. of Surgery, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

GRAND WARD ROUNDS – TRAUMA
Every Tuesday, 8 a.m. Arizona Medical Center, Tucson, AZ. Sponsor U of A. College of Medicine, Surgery Dept., Trauma Section Contact Martin Silverstein, M.D., U of A. College of Medicine, Tucson, AZ 85721. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

MONTHLY MEETING TUCSON ULTRASONOGRAPHY GROUP
First Wednesday of month, 4:30-5:30 p.m., Arizona Health Sciences Center. Sponsor: Dept. of Diagnostic Radiology. Contact: Irwin M. Freundlich, M.D., AHSC, Tucson, AZ 85724. Approved for 1 required hour per session toward ArMA CME Certificate.

TUCSON UROLOGICAL SOCIETY SCIENTIFIC MEETING
Monthly, Sponsor: Depts of Surgery & Urology, AHSC, Tucson. Contact: George W. Drach, M.D., U of A ealth Sciences Center, Tucson, AZ 85724. Approved for 1.5 required hours per session toward ArMA CME Certificate.

GENERAL UROLOGY CONFERENCE
Tuesday 5-6 p.m., Thursday noon-1:30 p.m. AHSC Meeting rms., & VA Hospital. Sponsor: Dept of Surgery/Urology Section. Contact: George W. Drach, M.D. Dept. of Surgery/Urology Section, U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour toward ArMA CME Certificate.

VA Hospital, Phoenix

CANCER SYMPOSIUM (fomerly Tumor Board)
Each Wednesday, 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact: Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

CARDIOLOGY CONFERENCE
2nd Thursday 1 p.m. T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D. VA Hospital, 7th & Indian School Rd. Phoenix, AZ Approved for required hours toward the ArMA Certificate in Continuing Medical Education

MEDICAL-SURGICAL CHEST CONFERENCE
1st and 3rd Thursday, 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

CLINICOPATHOLOGY CONFERENCE
4th Thursday of 3rd Mo. (Mar., June., Sept. & Dec.), 1 p.m., T-5. Sponsor VA Hospital Phoenix. Contact: Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ.

Approved for required hours toward the ArMA Certificate in Continuing Education.

DERMATOLOGY CONFERENCE

1st, 2nd & 3rd Wednesday Sponsor VA Hospital Phoenix. Contact. Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

HEPATOLOGY CONFERENCE

2nd and 4th Tuesday, 1 p.m., 2128. Sponsor: VA Hospital Phoenix. Contact: Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

GASTROENTEROLOGY CONFERENCE

1st and 3rd Tuesday, 1 p.m., T-5. Sponsor: VA Hospital Phoenix, Contact: Jasper L. McPhail, M.D. VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

GI CONFERENCE

(Special Program with U. of A. Consultants) 4th Friday, 1 p.m., T-5. Sponsor VA Hospital Phoenix Contact. Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

G.I.-RADIOLOGY CLINICAL CORRELATION CONFERENCE

Each Tuesday, 1 p.m., T-5. Sponsor: VA Hospital Phoenix. Contact: Jasper L. McPhail, M.D., Veterans Administration Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

MEDICAL SURGICAL G.I. CONFERENCE

1st & 3rd Friday - 2 p.m. -2128 Sponsor: VA Hospital, Phoenix. Contact: Jasper L. McPhail, M.D., VA Hospital, 7th Ave. & Indian School Rd., Phoenix. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL SERVICE GRAND ROUNDS

Each Friday, 11 a.m., T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

SURGERY-PATHOLOGY CONFERENCE

Each Thursday, 7 a.m., 2128 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

UROLOGY-PATHOLOGY CONFERENCE

Each Wednesday, 7 a.m., 2128 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th and Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

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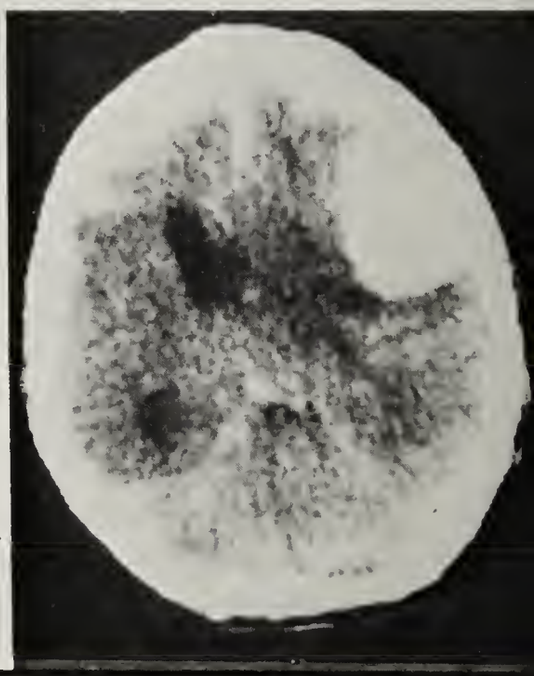
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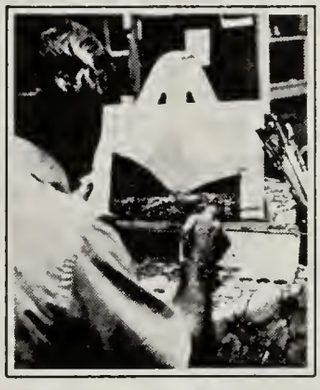
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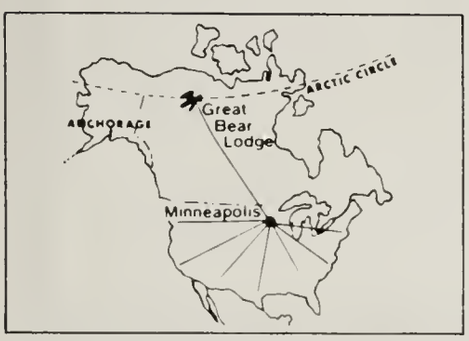
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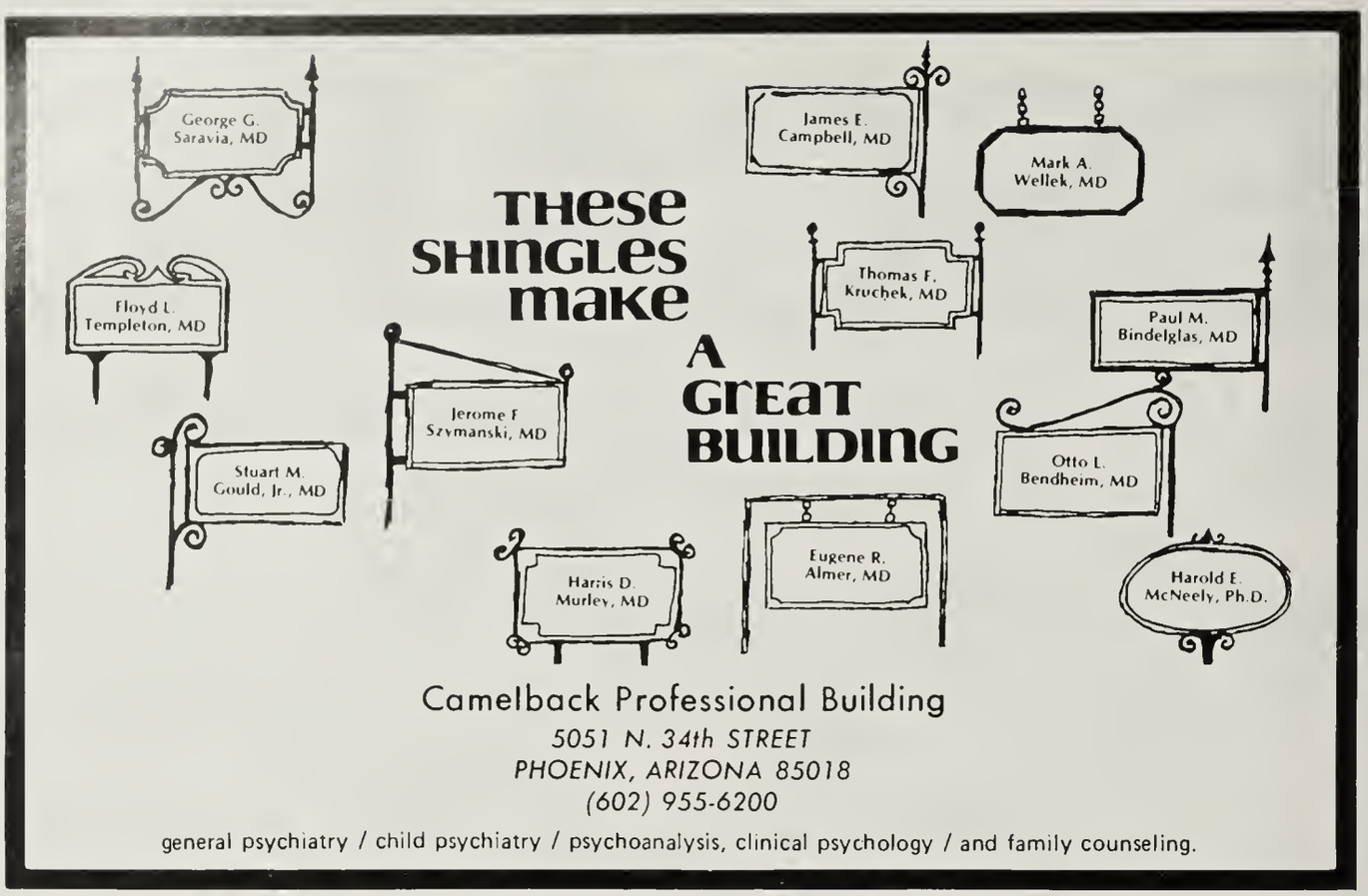
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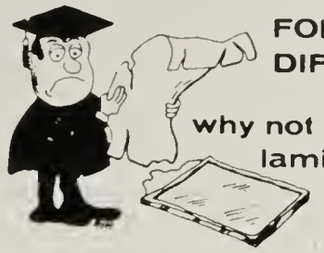
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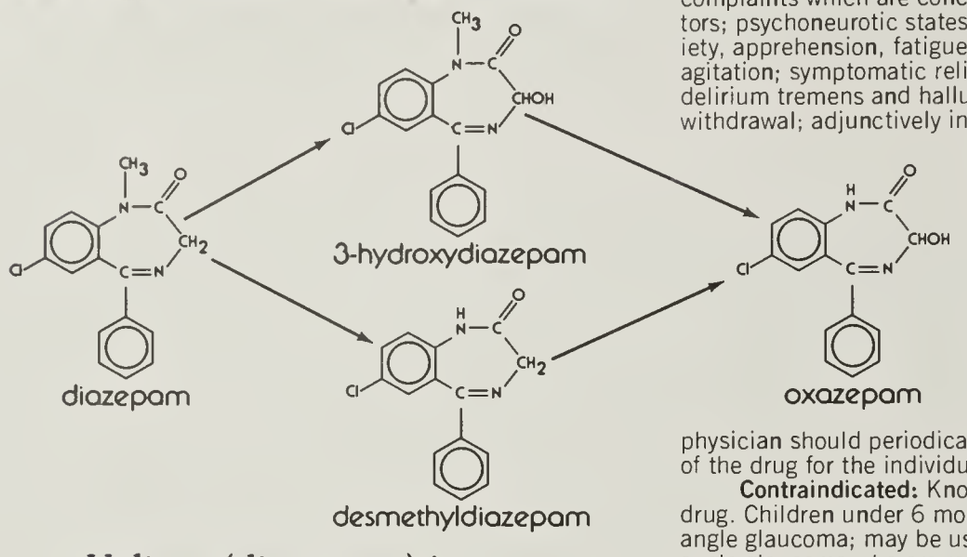
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Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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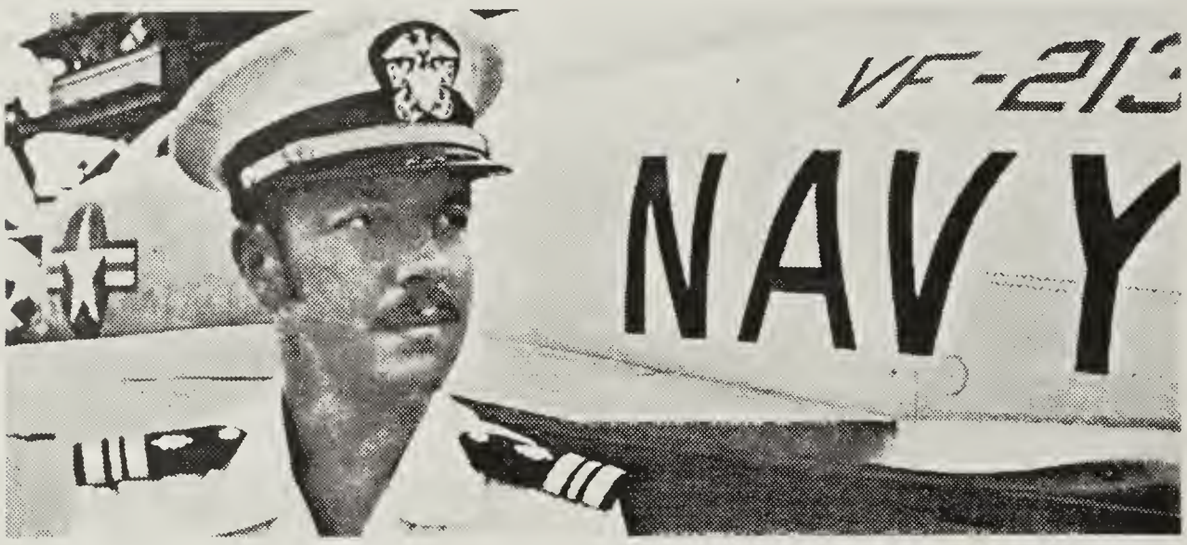
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Indications: Although the principal indication for cloxacillin sodium is in the treatment of infections due to penicillinase-producing staphylococci, it may be used to initiate therapy in such patients in whom a staphylococcal infection is suspected. (See Important Note below.)

Bacteriologic studies to determine the causative organisms and their sensitivity to cloxacillin sodium should be performed.

Important Note: When it is judged necessary that treatment be initiated before definitive culture and sensitivity results are known, the choice of cloxacillin sodium should take into consideration the fact that it has been shown to be effective only in the treatment of infections caused by pneumococci, Group A beta-hemolytic streptococci, and penicillin G-resistant and penicillin G-sensitive staphylococci. If the bacteriology report later indicates the infection is due to an organism other than a penicillin G-resistant staphylococcus sensitive to cloxacillin sodium, the physician is advised to continue therapy with a drug other than cloxacillin sodium or any other penicillinase-resistant semi-synthetic penicillin.

Recent studies have reported that the percentage of staphylococcal isolates resistant to penicillin G outside the hospital is increasing, approximating the high percentage of resistant staphylococcal isolates found in the hospital. For this reason, it is recommended that a penicillinase-resistant penicillin be used as initial therapy for any suspected staphylococcal infection until culture and sensitivity results are known.

Cloxacillin sodium is a compound that acts through a mechanism similar to that of methicillin against penicillin G-resistant staphylococci. Strains of staphylococci resistant to methicillin have existed in nature and it is known that the number of these strains reported has been increasing. Such strains of staphylococci have been capable of producing serious disease, in some instances resulting in fatality. Because of this, there is concern that widespread use of the penicillinase-resistant penicillins may result in the appearance of an increasing number of staphylococcal strains which are resistant to these penicillins.

Methicillin-resistant strains are almost always resistant to all other penicillinase-resistant penicillins (cross-resistance with cephalosporin derivatives also occurs frequently). Resistance to any penicillinase-resistant penicillin should be interpreted as evidence of clinical resistance to all, in spite of the fact that minor variations in *in vitro* sensitivity may be encountered when more than one penicillinase-resistant penicillin is tested against the same strain of staphylococcus.

Contraindications: A history of a previous hypersensitivity reaction to any of the penicillins is a contraindication.

Warning: Serious and occasionally fatal hypersensitivity (anaphylactoid) reactions have been reported in patients on penicillin therapy. Although anaphylaxis is more frequent following parenteral therapy it has occurred in patients on oral penicillins. These reactions are more apt to occur in individuals with a history of sensitivity to multiple allergens.

There have been well documented reports of individuals with a history of penicillin hypersensitivity reactions who have experienced severe hypersensitivity reactions when treated with a cephalosporin. Before therapy with a penicillin, careful inquiry should be made concerning previous hypersensitivity reactions to penicillins, cephalosporins, and other allergens. If an allergic reaction occurs, the drug should be discontinued and the patient treated with the usual agents, e.g., pressor amines, antihistamines, and corticosteroids.

Safety for use in pregnancy has not been established.

Precautions: The possibility of the occurrence of superinfections with mycotic organisms or other pathogens should be kept in mind when using this compound, as with other antibiotics. If superinfection occurs during therapy, appropriate measures should be taken.

As with any potent drug, periodic assessment of organ system function, including renal, hepatic, and hematopoietic, should be made during long-term therapy.

Adverse Reactions: Gastrointestinal disturbances, such as nausea, epigastric discomfort, flatulence, and loose stools, have been noted by some patients. Mildly elevated SGOT levels (less than 100 units) have been reported in a few patients for whom pretherapeutic determinations were not made. Skin rashes and allergic symptoms, including wheezing and sneezing, have occasionally been encountered. Eosinophilia, with or without overt allergic manifestations, has been noted in some patients during therapy.

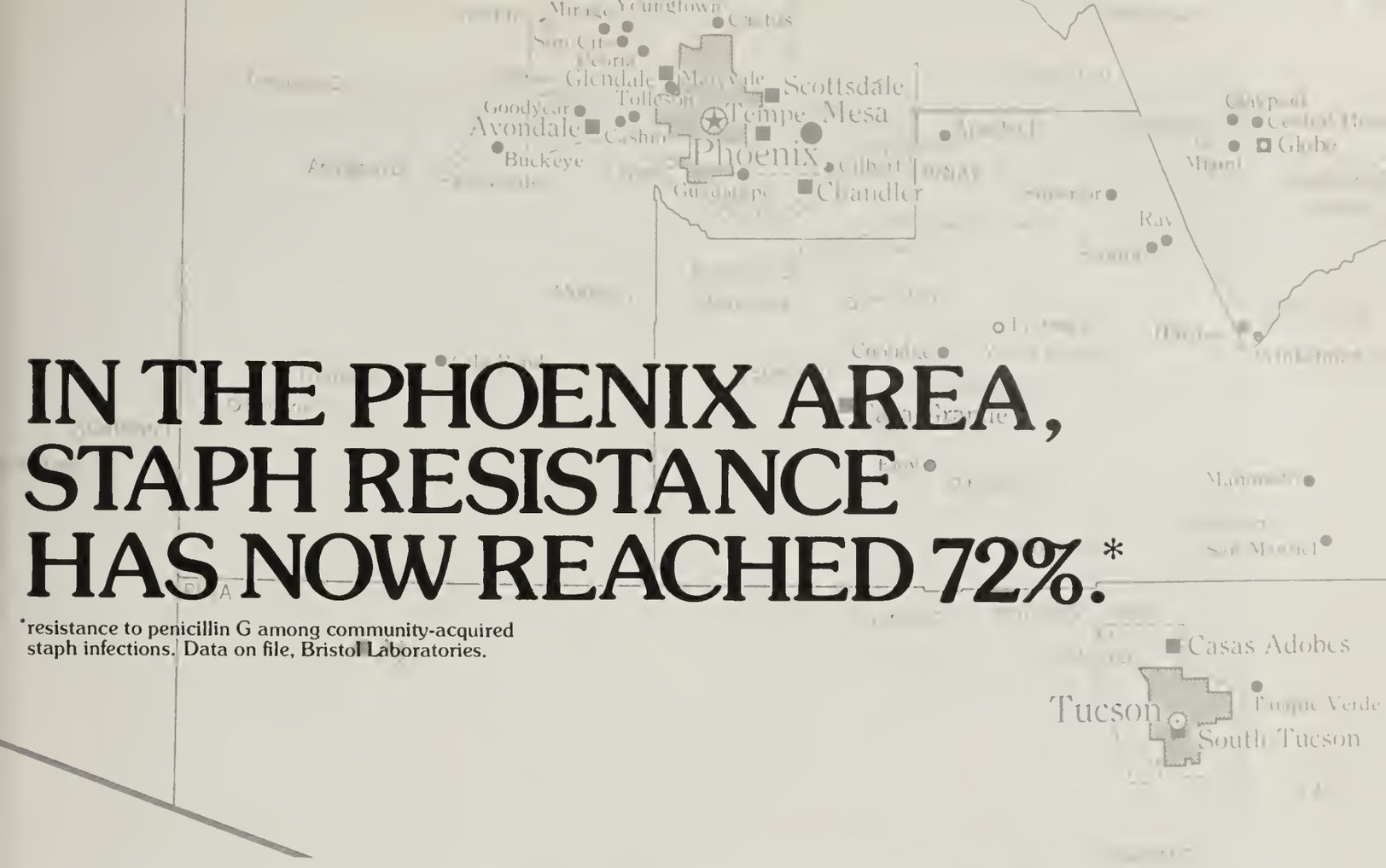
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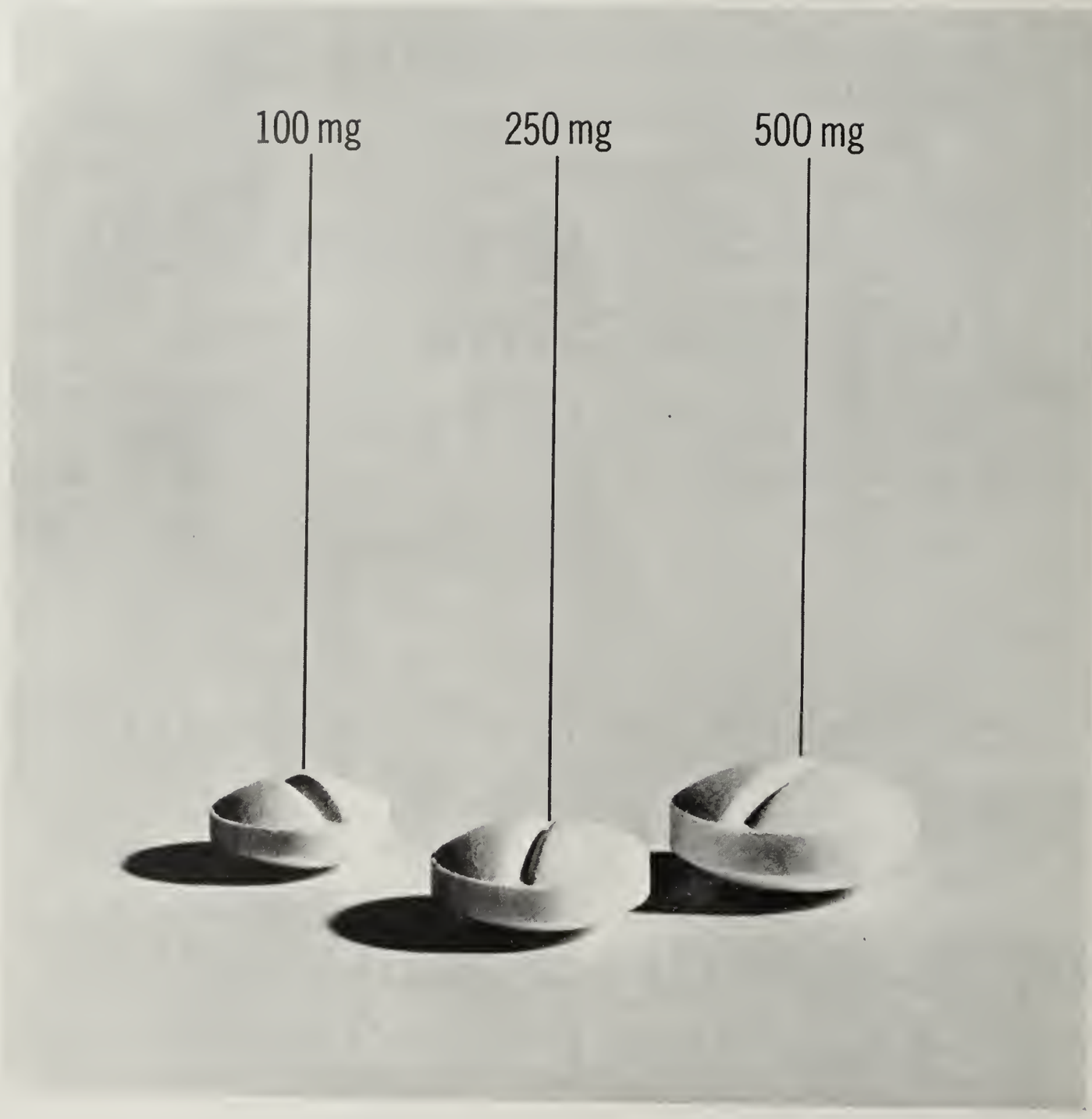
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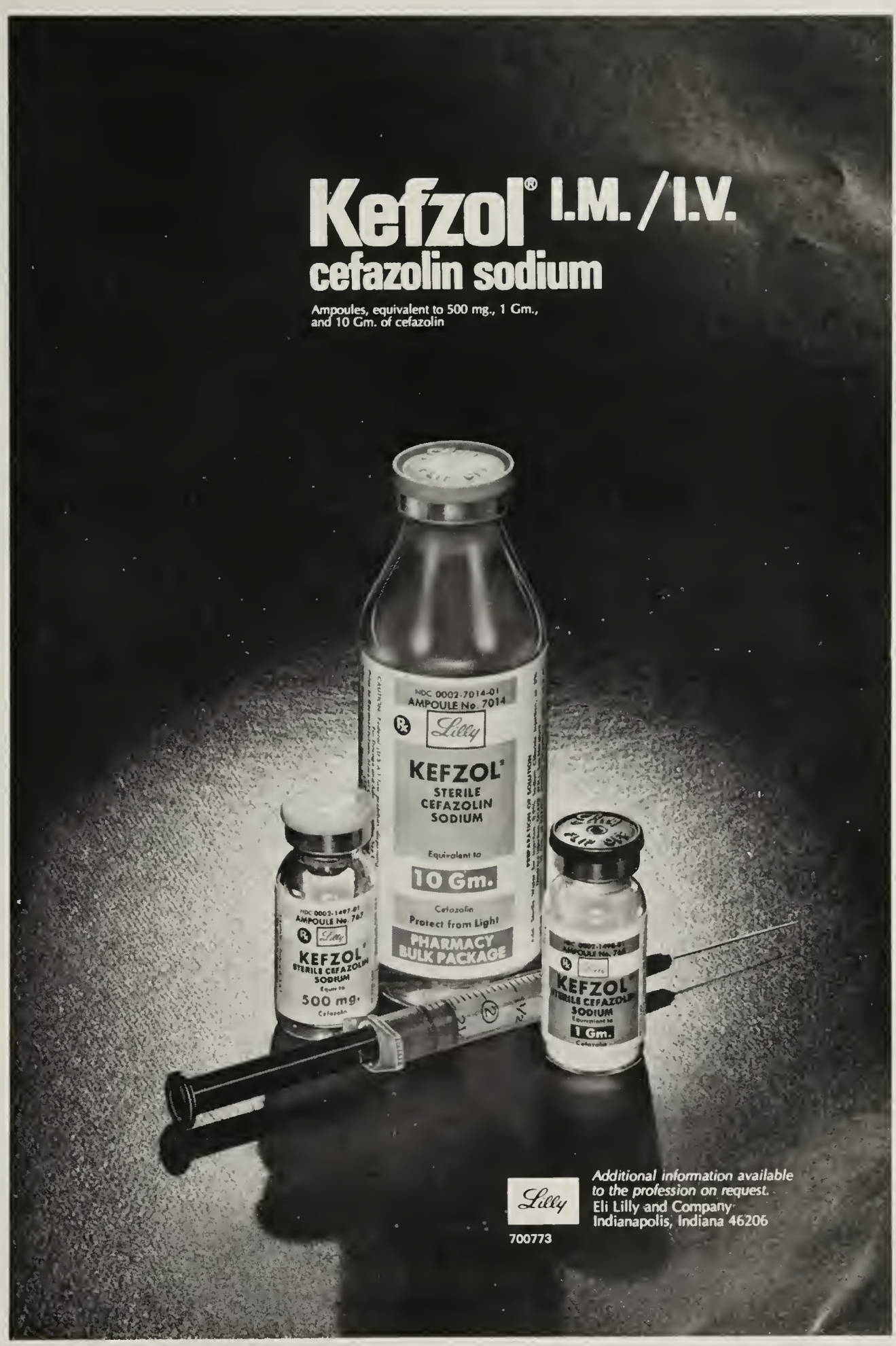
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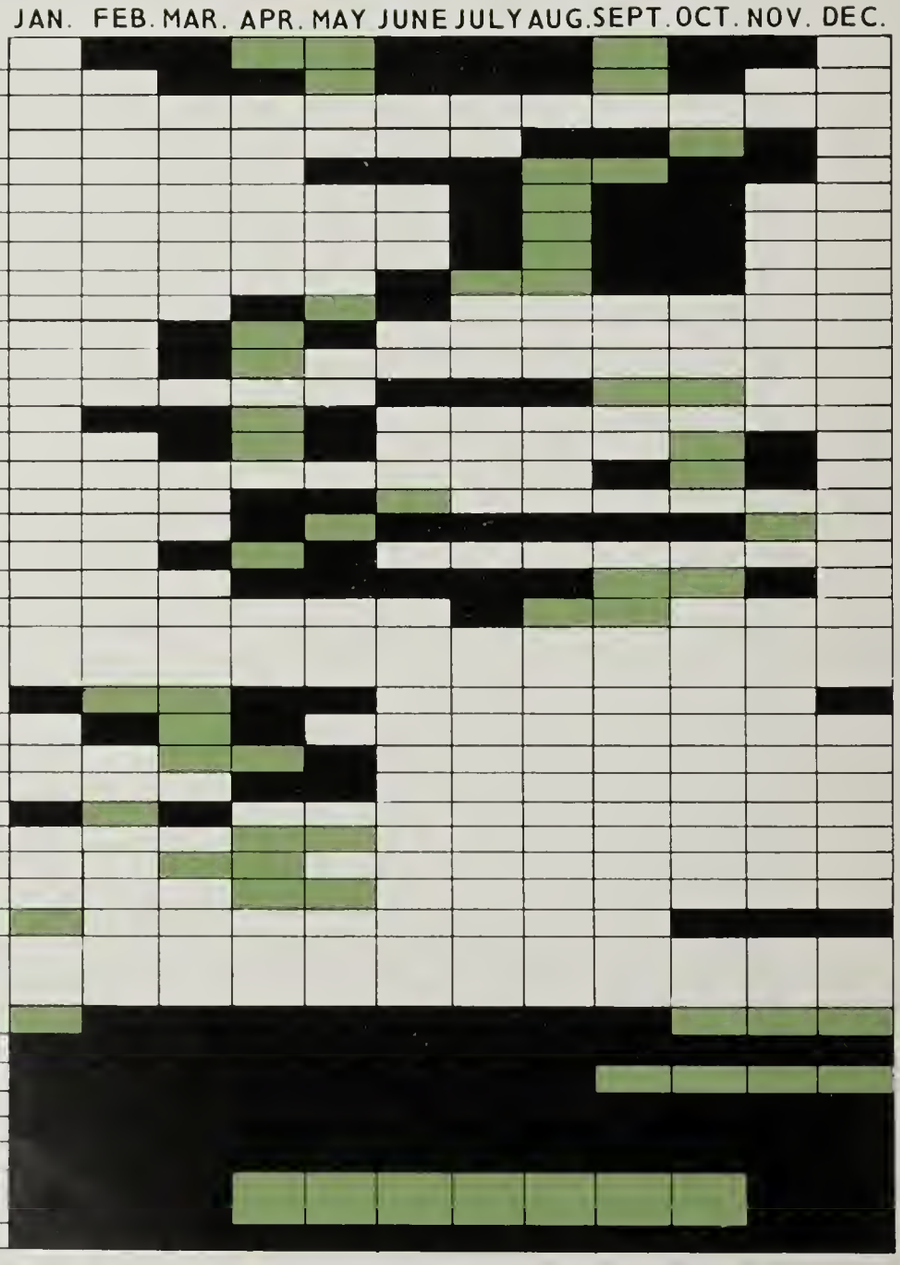
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SCIENTIFIC NAME	COMMON NAME
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2. Sorghum halepense	Johnson grass
HERBS & SHRUBS	
3. Allenrolfea occidentalis	Pickleweed
4. Amaranthus palmeri	Careless weed
5. Atriplex canescens	Wingscale saltbush
6. Atriplex lentiformis	Lens-scale saltbush
7. Atriplex polycarpa	All-scale saltbush
8. Atriplex wrightii	Annual saltbush
9. Beta vulgaris	Sugar beet
10. Chenopodium album	Lamb's quarter
11. Franseria ambrosioides	Canyon ragweed
12. Franseria confertiflora	Slender ragweed
13. Franseria deltoidea	Rabbit bush
14. Franseria dumosa	Desert ragweed
15. Hymenoclea monogyra	Burrobrush
16. Hymenoclea salsola	White burrobrush
17. Ricinus communis	Castor bean
18. Rumex crispus	Curly dock
19. Salsola kali	Russian thistle
20. Sarcobatus vermiculatus	Greasewood
TREES	
21. Cupressus arizonica	Arizona cypress
22. Fraxinus velutina	Arizona ash
23. Morus alba	Mulberry
24. Olea europaea	Olive
25. Populus fremontii	Cottonwood
26. Carya pecan	Pecan
27. Plantus spp.	Sycamore
28. Prosopis juliflora	Mesquite
29. Juniperus spp.	Juniper
FUNGI	
SPORULATION PERIODS	
70. Alternaria spp.	
71. Aspergillus spp.	
72. Helminthosporium spp.	
73. Hormodendrum spp.	
74. Penicillium spp.	
75. Rhizopus spp.	
76. Sphacelotheca holci (Johnson Grass Smut)	
77. Stemphylium spp.	

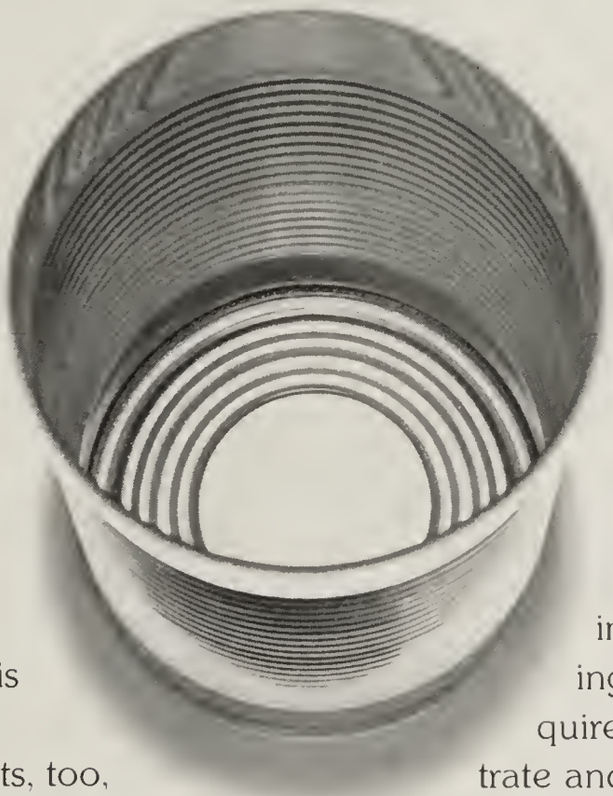


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Seminars in Chest Medicine

The Spectrum Of Cystic Fibrosis In Children And Adults

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INTRODUCTION

Cystic fibrosis (CF), an autosomal recessive disorder, remains the most common lethal genetic disease of Caucasian people. The diagnosis of CF is usually made based upon the clinical presentation of chronic respiratory and/or gastrointestinal symptoms, a positive sweat electrolyte test and, often, a family history of the disorder. Throughout the U.S. and Europe more than 50% of children are now diagnosed before 12 months of age. The vast majority of patients with cystic fibrosis in any age group present with primarily pulmonary (greater than 50%) and/or gastrointestinal (greater than 30%) symptoms. The manifestations of chronic cough, recurrent pneumonias, bronchiectasis with clubbing, pneumothorax, hemoptysis, and malabsorption secondary to pancreatic insufficiency are so well associated with cystic fibrosis that the diagnosis is usually not delayed.

However, it is often not appreciated by physicians that CF may be associated with a broad spectrum of less common manifestations as well as extreme variability in severity of illness. Lack of familiarity with these manifestations may delay diagnosis until adolescence or adulthood.

We would like to acquaint the practitioner with the various manners of presentation of patients with CF, emphasizing the more subtle aspects of the disorder (Table I). It is important that CF be diagnosed early in life so that appropriate genetic counseling can be given and maintenance and preventive therapies instituted.

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85 Beckerman is a recipient of clinical fellowship grant from the
Amer ng Association.

NEONATAL PERIOD (TABLE 1)

Approximately 10-15% of children with CF present during the neonatal period with intestinal obstruction secondary to *meconium ileus* or *meconium peritonitis* following prenatal intestinal perforation. Variants of this manifestation which may not be well appreciated include radiographic *intra-abdominal* and *scrotal calcifications*, *small bowel atresias* (25% of all such atresias are associated with CF) and the *meconium plug syndrome*. Persistent *cholestatic* (*obstructive*) *jaundice* may be a primary manifestation of CF in the neonatal period. The presumed pathogenesis of the increase in direct bilirubin is inspissation of abnormal mucous secretions within the intrahepatic and extrahepatic bile ducts. The jaundice which is of moderate severity and subsides spontaneously within the first six weeks of life, presents the clinician with more of a diagnostic than a treatment dilemma. No therapy is required.

INFANCY (TABLE I)

Abnormal electrolyte excretion (elevated sodium, chloride and potassium concentrations) by eccrine sweat glands occurs in virtually all CF patients of any age group. The sweat rate, however, is normal in CF patients. The abnormal concentration of salt deposited on the infant's skin has prompted many parents of children with CF to seek medical consultation because of *"salty kisses,"* or salt crystals precipitating on eyebrows. A valid sweat test

must be accomplished using a reproducible quantitative technique, such as the pilocarpine iontophoresis test. At least 75-100 mgm of sweat should be collected in duplicate samples. A sweat chloride concentration of greater than 60 mEq/liter in children and greater than 75 mEq/liter in adults is diagnostic of CF. A quantitative collection may be difficult to achieve in the small infant whose sweat rate only gradually increases to normal levels by two to four months of age. A minority of CF patients may fall into a "grey zone" area of sweat chloride concentration (50-60 mEq/liter for children and 60-75 mEq/liter for adults) so that a tentative diagnosis of CF may have to be based upon clinical and/or genetic factors. False negative sweat tests usually reflect lack of sufficient quantity of sweat but have been seen occasionally in CF infants who have hypoproteinemia and edema. False positive tests occur with fair frequency (3-5%) with qualitative sweat testing techniques.

As a result of tremendous sweat losses of sodium, chloride and potassium, CF patients are predisposed to *acute heat prostration* with hypotension. This may be seen rarely during winter months in infants who have a low intake of dietary sodium chloride, are predisposed to high fevers and who cannot easily conserve sodium and water. It is more commonly seen during summer months, but in arid climates, like Arizona, may occur throughout the year. Prophylactic salt supplementation is required to prevent this complication. Undiagnosed infants with CF may develop *hypoelectrolytemia and metabolic alkalosis* during any season. A recent review of CF Center records at the Arizona Health Sciences Center revealed that between 1972-1977, five of ten (50%) CF infants diagnosed between one and twelve months of age initially presented with a syndrome of hyponatremia, hypochloremia and hypokalemia with moderately severe metabolic alkalosis. None of these infants exhibited more than five percent clinical dehydration and acute gastrointestinal losses appeared to be of insufficient magnitude to explain their electrolyte disturbances. It is postulated that acute fluid and electrolyte losses due to mild gastrointestinal or respiratory illness when superimposed upon chronic sweat electrolyte depletion may result in metabolic alkalosis.

An occasional CF infant may present with a picture of *failure to thrive* associated with *hypoproteinemia, generalized or peripheral edema* and *anemia.* The hypoproteinemia is secondary to malabsorption or poor utilization of dietary proteins (eg. soy formulas) or to breast milk because of its relatively low protein content. The etiology of the accompanying anemia cannot be explained on blood loss or iron deficiency. It has been suggested that there may be a *hemolytic process* associated with *vitamin E deficiency* but this has not been proven. Overt *hemorrhage* secondary to *hypoprothrombinemia* may occur in CF infants as well as older patients. Several factors seem to be responsible for the depletion of vitamin K dependent factors: (a) malabsorption of fat soluble vitamins (D, E, K, A); (b) use of continuous broad spectrum antibiotics which eliminate the normal intestinal flora responsible for endogenous vitamin K production; and (c) severe liver disease (rarely).

TABLE I

MANIFESTATIONS OF CYSTIC FIBROSIS (CF)

AGE GROUP	MANIFESTATION	OVERALL INCIDENCE
Neonatal	Meconium ileus; meconium peritonitis; intestinal atresia	10-15%
	Obstructive jaundice	rare
	Salty taste	nearly 100%
	Heat exhaustion	occasional
Infancy	Metabolic alkalosis	unknown
	Hypoproteinemia, edema and anemia triad	rare
	Hypoprothrombinemia and hemorrhage	rare
	Increased intracranial pressure (hypovitaminosis A)	rare
	Hydrocele and inguinal hernia	rare
	Rectal prolapse	20-25%
	Intussusception	1-2%
	Nasal polyps	10-15%
Toddler-later Childhood	Chronic secretory otitis media	30-40%
	Biliary cirrhosis/ portal hypertension	2%
	Pansinusitis	greater than 90%
and	Acute pancreatitis	less than 10%
	Infertility in males	98%
	Infertility in females	unknown
Adolescence-Adulthood	Diabetes mellitus requiring insulin	1-2%
	Meconium ileus equivalent	15%
	Chronic cough; recurrent pneumonias; bronchiectasis	nearly 100%
All age groups	Pancreatic insufficiency with malabsorption	85-90%
	Cor pulmonale	75-100%

TODDLER, CHILDHOOD (TABLE I)

Rectal prolapse is a relatively common complication of CF in infants and toddlers between nine months and five years of age. Cystic fibrosis is the most common distinct entity causing rectal prolapse in North America and any child with such a prolapse should have a sweat test.

Rectal prolapse may be the presenting manifestation of CF in 5% of children. The predisposing factors seem to be the poor overall nutritional status, decrease in perianal subcutaneous tissue and muscle mass, frequent bulky bowel movements, and chronic severe cough with increased intra-abdominal pressures. Surgical correction is rarely required because manual reduction, dietary manipulation and control of respiratory symptoms usually prevent recurrences.

The incidence of respiratory allergy is probably not increased in CF patients. *Sinusitis* occurs in nearly all patients. The incidence of *nasal polyposis* is 10-15%. The polyps probably are due to infection, glandular obstruction, and, in some cases, coexistant allergies. Since nasal polyps probably occur more commonly in CF children than in allergic non-CF children, any child with nasal polyps should be evaluated for CF with a sweat test.

Recurrent *intussusception* may be a rare manifestation of CF (1%). Idiopathic intussusception in childhood most commonly occurs between 5 and 8 months of life and has classic presenting signs and symptoms. The incidence of intussusception due to CF peaks between 7 and 9 years of age and is usually of the ileocolic type. Whereas surgical reduction used to be required in the majority of episodes, that form of treatment has been replaced by medical reduction using Hypaque® or Gastrografin® enemas.

Although focal biliary cirrhosis is commonly seen histopathologically (50-75%) at autopsy, clinical liver disease in the form of *cirrhosis with portal hypertension* and bleeding esophageal varices is rarely seen in CF (2%). Advanced cirrhosis usually is associated with chronic pulmonary and gastrointestinal manifestations of CF but may rarely be a presenting manifestation, making CF part of the differential diagnosis of early onset biliary cirrhosis.

It is not unusual to feel hard *fecal masses* especially in the right lower quadrant of CF children and adults. They usually cause no symptoms but an occasional patient will complain of acute transient abdominal pain. The fecal masses may disappear or persist for months. They may serve as "lead points" for intussusception or lead to bowel obstruction which is called *meconium ileus equivalent*. The latter presents with cramping abdominal pain and is associated with vomiting, decreased or absent bowel movements, abdominal distention and large fecal masses on examination. The etiology is uncertain but probably relates to abnormal fecal material and intestinal mucus.

ADOLESCENCE, ADULTHOOD (TABLE I)

The older patients who have escaped the diagnosis of CF throughout childhood may be more difficult to recognize. They usually carry other diagnoses such as asthma, chronic sinusitis, bronchiectasis, chronic bronchitis, or celiac-sprue. Quite often, they have had extremely mild respiratory disease and very mild or no gastrointestinal manifestations. Adults are usually diagnosed because of manifestations which are rarely seen in children with CF.

Episodes of *acute pancreatitis* are usually seen in patients with nearly normal pancreatic function. This rare presentation is usually associated with the 10-15% of CF patients who do not demonstrate chronic pancreatic insufficiency. They usually experience excellent growth and development throughout childhood and adolescence.

Bleeding esophageal varices secondary to *progressive biliary cirrhosis* with *portal hypertension* has been reported as the only obvious manifestation of CF in adolescents and adults. This is a rare occurrence. *Meconium ileus equivalent* may occur in 15% of CF adults. Although *carbohydrate intolerance* (chemical diabetes) increases with advancing age in CF (40-60%), only 1-2% eventually

TABLE II

CLINICAL MANIFESTATIONS REQUIRING CONSIDERATION OF CYSTIC FIBROSIS

RESPIRATORY
- Chronic loose cough
- Pulmonary atelectasis
- Recurrent pneumonia
- Refractory bronchial asthma
- Staphylococcus aureus pneumonia/empyema
- Bronchiectasis
- Digital clubbing/hypertrophic pulmonary osteoarthropathy
- Nasal polyps
- Chronic pansinusitis
- Unexplained cor pulmonale

GASTROINTESTINAL
- Neonatal small bowel atresia
- Diffuse peritoneal calcifications
- Neonatal obstructive jaundice
- Failure to thrive
- Steatorrhea
- Chronic diarrhea
- Late onset intussusception; any mechanical obstruction (meconium ileus equivalent)
- Rectal prolapse
- Unexplained cirrhosis, portal hypertension
- Hypoproteinemia, anasarca

MISCELLANEOUS
- "Salty kisses"
- Hemorrhage secondary to vitamin K deficiency
- Increased intracranial pressure secondary to hypovitaminosis A
- Heat exhaustion (acute salt depletion)
- Metabolic alkalosis and hypoelectrolytemia
- Aspermia

manifest overt *chemical diabetes* requiring insulin treatment. The diabetes of CF is unlike the genetic type in that there is a mechanical obstruction to insulin (and possibly glucagon) secretion into the blood stream in CF because of the severe pancreatic fibrosis. CF patients with diabetes rarely develop ketoacidosis.

Cystic fibrosis may be diagnosed in the adult who seeks medical advice for evaluation of *infertility*. Reproductive failure is almost universal in CF males (98%). The ejaculate is low in volume and contains no sperm. Obstruction of the epididymis and vas deferens has been implicated in the pathogenesis of male infertility in CF. The endocrine function of the testes (as well as other endocrine glands) is normal. The incidence of infertility in CF females is not exactly known but reproductive failure is common. Its cause is presumably related to mechanical blockade of sperm motility by abundant and viscid vaginal secretions. The acquisition of secondary sexual characteristics are usually delayed in CF patients who have severe pulmonary disease and poor nutritional status.

It is clear in reviewing several recent reports that CF patients may escape diagnosis until adolescence or even adulthood. The reasons for this phenomenon may be two fold. First, there seems to be mild clinical expression of the disorder in some patients (genetic variability). Second, the subtle manifestations of CF are not widely appreciated by physicians in practice. In fact, CF may be considered a *"great masquerader"*. It has been estimated, based on Caucasian birth rates and the number of known CF patients, that at least 75-100 patients with CF have not yet been diagnosed in Arizona. Table II provides a differential diagnosis of CF. Recognition of the major and minor manifestations of CF and the accessability of accurate and reliable sweat tests, makes it possible to diagnose CF in patients of any age.

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Emotional Disorders

Expectations

William B. McGrath, M.D.

The mind is our vital link between the past and the future. What follows? The most constant and important function of intelligence is anticipation. And to an unrecognized degree one's expectations predict and control one's behavior.

This sequence — past experience, expectation, future behavior — can be observed in nearly all purposeful activities. The successful boxer or tennis player anticipates his opponent's next move and acts accordingly. The physician or surgeon who has learned what to expect will know what to do or what not to do.

Any individual has had innumerable experiences in which there have been casual or at least coincidental or reciprocal relationships. One's experiences are the source of his expectations. "If I will cry someone will comfort me." "If I am quiet no-one will take hostile notice of me." Even one's dress will be determined to a significant degree by memories of rejections or acceptances, punishments or rewards of infinite variety in kind and intensity.

The easiest way to understand a person is to observe and come to recognize his expectations. Naturally they will have undergone changes: Some will have been reinforced by success; some will have been more or less dampened by failure.

To influence a person is to modify his expectations and thereby change his behavior. This requires a special perspective and a learned technique.

Events which conditioned the individual's expectations will have been different from your own. Matters wherein the contrast is too great should not be brought into premature consideration. You simply cannot speak the same language of expectations. You cannot find early and mutual empathy, for example, with a patient who was orphaned and abused as a child. The gulf is too wide. Similarly, a great actor or lawyer or surgeon might be a helpful mentor to a fairly competent colleague but not to an unskilled amateur.

At the nearer end of the spectrum, if your biographies are very similar you can anticipate little or no change or need of change; e.g. if you have always shared the same fundamentalist beliefs.

From: 5055 N. 34th St., Phoenix, AZ 85018.

Rapport and effective influence begin only in an intermediate zone in which you and the other person have something but not too much in common.

With you at his side the patient trudges back down the long and twisting road of his experiences. He remembers and recounts significant events and how he reacted to them. You ask: Looking back, was your reaction appropriate? Was it perhaps histrionic or passive-aggressive or paranoid? How might it have been different? In view of what you have learned, might you have been less judgemental and without fear or bitterness?

Such a review of the past gradually emerges into a rehearsal of the future. Faced with similar sets of circumstances or conflicts, the patient will have a choice of wiser and more enriching methods of coping. When a person can be taught to evaluate his past (which is himself) more philosophically, then his expectations will change — and so will he.

Seminars in Endocrinology and Metabolism

Cushing's Syndrome Or "Cushingoid Obesity": How To Make The Distinction

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Cushing's syndrome is a rare disease that occurs mostly in women between the second and sixth decades of life. The typical clinical features of Cushing's syndrome are usually due to an excess of endogenous cortisol or exogenous glucocorticoids (Table 1). Occasionally the signs and symptoms of excessive adrenal androgens or mineralocorticoids may be more prominent than those related to the glucocorticoids; but regardless, the excessive production of glucocorticoids remains the hallmark of the disease. The four causes of Cushing's syndrome are: iatrogenic or excessive exogenous glucocorticoids, Cushing's disease or a disorder of the hypothalamic-pituitary-adrenal (HPA) axis resulting in bilateral adrenal hyperplasia, primary adrenal adenomas or carcinomas, and excessive stimulation of the adrenals by ACTH produced by ectopic tumors. Although Cushing's syndrome is rarely encountered in most general medical practices, "Cushingoid obesity" or patients suspected of having hypercortisolism is not, and effective screening is essential for early diagnoses. The tests used to make the distinction are available to most physicians and should be utilized by them. The following discussion will outline the use of these and other adrenal function tests to both screen for and make the diagnoses of Cushing's syndrome in the Cushingoid obese.

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In normal, unstressed individuals, cortisol regulates its own secretion through the hypothalamic-pituitary-adrenal (HPA) axis. Corticotropin—releasing factor (CRF) elaborated by the hypothalamus induces a release of adrenocorticotropic hormone (ACTH) from the pituitary which inturn leads to production and release of cortisol from the adrenal cortex. Plasma cortisol has a negative feedback relationship with CRF, and hence with ACTH. Through the HPA axis, cortisol production is closely regulated and does not exceed metabolic demands. During times of stress, cortisol production is increased to allow the appropriate metabolic adaptation to take place and CRF release becomes autonomous from plasma cortisol concentrations. These same biochemical changes in stress are found in patients with Cushing's syndrome, namely, adrenocorticol hyperfunction which is independent of the feedback regulation by cortisol. Since both stress and Cushing's syndrome are accompanied by an increased production and secretion of cortisol which cannot be suppressed normally with glucocorticoids, adrenal function studies to diagnosis Cushing's disease should not be done during periods of either emotional or physical stress. Conversely, patients in whom adrenal function studies are abnormal, stress should be ruled out as the cause of the abnormality.

The HPA axis becomes most active shortly after the onset of sleep resulting in a surge of ACTH from the

TABLE 1:
7 Cardinal manifestations of Cushing's Syndrome

1. Plethora, moon face	90%
2. Truncal obesity	85%
3. Glucose intollerance	85%
4. Hypertension	80%
5. Osteoporosis	70%
6. Amenorrhea, hypogonadism	70%
7. Purple striae, ecchymosis	60%

pituitary, reaching a peak in eight hours, and falling the remainder of the day. The result, cortisol at 8:00 a.m. is usually between 10 to 25 mcg% declining to less than 50% by 8:00 p.m. Since this diurnal variation of cortisol is usually lost in adrenocorticol hyperfunction, a measurement of the evening plasma cortisol is more apt to give an elevated value than is the morning cortisol concentration. As a general rule in diagnosing endocrine diseases, suppression studies during excessive hormone production and stimulation studies during insufficient hormone production usually give a clearer separation of normal from abnormal than does the measurement of absolute concentrations. This concept applies to the diagnosis of Cushing's syndrome as well. One mg of dexamethasone given at 11:00 p.m. with measurement of the plasma cortisol the following morning is an excellent screening test. Normally the plasma cortisol suppresses to less than 6 mcg%. Only rarely is this test normal in Cushing's syndrome. Unfortunately, a failure to normally suppress does not establish the diagnosis of Cushing's syndrome, and many times it is the "Cushingoid obese" patient that will have a false positive test. In those patients in whom the overnight dexamethasone suppression test is abnormal, a 24 hour urine collection for urinary "free" cortisol and creatinine (to ensure an adequate collection) will more clearly distinguish Cushing's syndrome from normal. The urinary "free" cortisol takes advantage of the fact that a certain fraction of the plasma free cortisol or metabolically active form of cortisol will be secreted into the urine and when measured as such gives a very accurate measurement of total cortisol production. Because urinary 17-hydroxy-corticosteroids (17-OHCS) represents a metabolic degradation product of cortisol and measures only about 30-50% of the total cortisol production, there is considerable overlap between normal and abnormal, and should not be used as a screening test. Its measurement is of greater value when used in conjunction with a 2 and 8 mg dexamethasone suppression test that will be discussed latter. Although the urinary "free" cortisol is an excellent screening test, the difficulty in obtaining complete urine collections on outpatients makes the overnight dexamethasone suppression study a more practical test to perform.

Patients who fail to suppress following 1 mg of dexamethasone and who have an elevated urinary "free" cortisol, should be admitted to the hospital for further clinical evaluation and further testing of adrenal function. In those patients who are strongly suspected of having Cushing's syndrome despite a normal 1 mg suppression study or in those with high normal urinary "free" cortisol determinations, the tests should be repeated at three to six month intervals to be certain that one is not missing early adrenocortical hyperfunction. In the patients with abnormal screening studies, the formal 2 and 8 mg dexamethasone suppression studies should be done to distinguish those patients with Cushing's disease from the patients with adrenocortical tumors or ectopic ACTH production. All patients with Cushing's syndrome fail to suppress with the 2 mg study, confirming the abnormal screening tests, but only patients with Cushing's disease or over-activity of the HPA axis will suppress to less than 50% of the baseline of urinary "free" cortisol or 17-OHCS with high dose dexamethasone; whereas, patients with adrenocortical tumors or ectopic ACTH secretion fail to suppress even with the high dose dexamethasone since their cortisol production is completely independent of the HPA axis. The distinction between adrenocortical tumors and ectopic production of ACTH can be made by ancillary adrenal and radiographic studies. A radiographic search for a malignancy, measurement of the serum ACTH concentration, adrenal radiography, and soon to be more widely available adrenal scanning with special radionucleotides will enable a clear distinction between these two etiologies of excessive cortisol production and will prevent the embarrassing situation of finding bilateral adrenal hyperplasia at operation only latter to discover an occult malignancy that was secreting ACTH.

In summary, patients with "Cushingoid obesity" can be effectively screened for adrenal disease as outpatients with the overnight dexamethasone suppression test and when necessary, measurement of the urinary "free" cortisol. These same tests can be used to follow those patients who are suspected of having Cushing's syndrome but have borderline test results. In those with abnormal tests, hospitalization with additional adrenal function studies to confirm the diagnosis and determine its etiology must be done. Although Cushing's syndrome is a rare disease, "Cushingoid obesity" is not, and a distinction between them often must be made by the appropriate adrenal function studies. Early diagnosis and successful treatment of states of excessive cortisol production is rewarding to the physician and most importantly, can have a dramatic improvement in the patient's quality of life.

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Seminars in Gastroenterology and Liver Disease

Evaluation And Treatment Of The Acute Attack Of Ulcerative Colitis

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Although the treatment of ulcerative colitis is far from satisfactory, a number of modes of therapy are available. This paper will discuss the evaluation of the acute attack of ulcerative colitis and the treatment of that attack.

Ulcerative colitis can be a dangerous, life-threatening disease both in the short term from massive bleeding or toxic dilatation or, late in the course of the disease, from carcinoma of the colon or one of the rare long term complications such as liver failure. It has been my experience that the acute attack of colitis is frequently underestimated from the standpoint of its severity. As a consequence, the acute attack of colitis is commonly not treated appropriately because the gravity of the patient's condition is not recognized.

EVALUATION OF THE ACUTE ATTACK OF COLITIS

Outlined in Table I are criteria for the evaluation of the acute attack of ulcerative colitis. These criteria have important prognostic and treatment implications.[1] What should impress the physician is what may be considered minimal symptomatology in other gastrointestinal illnesses (for example, viral gastroenteritis) may be a manifestation of a severe attack of ulcerative colitis. Prior to specific therapy for ulcerative colitis, i.e.: steroids and Azulfidine® , the mortality rate for patients admitted to the hospital with severe ulcerative colitis was approximately 30%.[2]

TABLE I

CRITERIA FOR EVALUATING AN ACUTE ATTACK OF ULCERATIVE COLITIS*

SIGNS/SYMPTOMS	SEVERITY OF ATTACK		
	Mild	Moderate	Severe
Diarrhea (stools per day)	4 or less	4 to 7	7 or more
Macroscopic blood	Usually not	Usually	Yes
Fever	None	±	99.5° F mean (PM)
Tachycardia (per minute)	No	~ 90	> 90
Anemia (75% normal)	No	±	Usually
Erythrocytic sedimentation rate (30 mm/per hr)	Usually not	Usually	Yes

*Adapted from Truelove and Witts[1]

From: 1616 E. McDowell, Suite 204, Phoenix, AZ 85006.

Anyone with massive hemorrhage in the colon or radiographic evidence of toxic dilatation of the colon should be placed in the category of severe ulcerative colitis. However, patients can be severely ill without severe bleeding or dilatation. Recognition of the patient with ulcerative colitis who is moderately severe or severely ill and initiation of early supportive and specific therapy can be expected to decrease the mortality rate significantly.

When a patient does not have a prior history of ulcerative colitis the differential diagnosis can be difficult. The differential diagnosis in Table II presents the majority of conditions which may present with abnormal sigmoidoscopic examination. Some of these are easily differentiated, either historically or on the appearance of the sigmoidoscopic examination. However, there is nothing pathognomonic about the sigmoidoscopic examination in ulcerative colitis and the differential in the patient with an acute attack may be very difficult. The most important differential is the exclusion of amebiasis which should be done through the examination of wet preps of the stool, specimens submitted to the laboratory in polyvinyl alcohol and formalin and by obtaining an IHA titer for amebiasis.

Sigmoidoscopy is the most important examination when evaluating patients for ulcerative colitis. Controversy exists as to whether a barium enema should be performed. My personal bias is that a barium enema may make the patient very ill and that it should be avoided during the acute attack of colitis. Information obtained at sigmoidoscopy is usually adequate for treatment in the acute phase.

TREATMENT OF THE ACUTE ATTACK OF ULCERATIVE COLITIS

MILD ATTACK

The mild attack of ulcerative colitis can usually be treated on an outpatient basis. The patient should be given detailed instructions to return if the illness becomes more severe. Salicylazosulfapyridine (Azulfidine®) should be given in doses of 4-6 gm/day rather than the usual maintenance dose. The physician must remember, however, that side effects (headaches, fever, gastrointestinal upsets) are common with doses in this range and will need to be differentiated from symptoms associated with the primary illness.

Systemic steroids are used to variable degrees in the acute exacerbation. One can obtain almost any guideline he wishes in the literature. My personal policy is not to treat mild disease with the use of systemic steroids. However, if the attack is prolonged or responds poorly, prednisone, 10 mg, b.i.d., will usually interrupt the process quickly. If the patient is allergic to Azulfidine® or is unable to tolerate adequate dosage because of side effects this would be another indication to initiate systemic steroid therapy early.

Rectal steroids (e.g., one Cortenema® , b.i.d.) are very useful in mild attacks. They are particularly helpful in alleviating symptoms of urgency and tenesmus.

TABLE II

DIFFERENTIAL DIAGNOSIS

1. Ulcerative proctitis
2. Granulomatous colitis
3. Amebiasis
4. Bacillary dysentery
5. Ischemic colitis
6. Pseudomembranous colitis
7. Gonorrhea proctitis
8. Radiation colitis
9. LGV
10. Other systemic disease
 a. Hemolytic-uremic syndrome
 b. Henoch-Schönlein's purpura
 c. Behc,et's syndrome

MODERATE ATTACK

The moderate attack is more dangerous than one would predict by the initial clinical appearance of the patient. These patients are best treated in the hospital. They are much more likely to have nutritional and fluid and electrolyte problems than the patients with milder forms of the disease.

Corticosteroids should be used. Although it is difficult to generalize, the typical patient with a moderate attack of colitis might be started on 40 mg. of prednisone daily in divided doses. Tapering of the daily dosage will depend upon response. Usually the response is rather prompt and I have found no difficulty in tapering the patients rapidly over a three to four week period. In others, however, it may be necessary to decrease the steroids very slowly over a period of several months. It is difficult to draw a line beyond which steroids should be abandoned in favor of colectomy. However, the physician must remember that he is dealing with a curable disorder and that prolonged high doses of prednisone should not be used.

Salicylazosulfapyridine and rectal steroids may be used as indicated in a mild attack.

SEVERE ATTACK

The severe attack should be treated in the hospital. Fluid and electrolyte abnormalities are common in these patients (hypokalemia, hypoalbuminemia) and may need correction since they may potentiate the seriousness of the condition and predispose the patient to megacolon. Anemia is usually present. Whereas one might not transfuse a patient with one or two units of blood to bring his hematocrit to normal in other clinical situations, it is generally recommended that patients with ulcerative colitis be transfused to the normal range if they are seriously ill. Transfusions often seem to bring about substantial improvement in the patient's course beyond what one would expect from the increase in hemoglobin.

Depending upon the gravity of the attack, one may keep the patient on a nothing-by-mouth (NPO) regimen or may feed a low residue, high calorie, high protein diet. Milk should be removed from the diet because of the

apparent potential for aggravation of colitis with lactose intolerance. In addition, there seems to be a higher incidence of lactose intolerance among patients with ulcerative colitis.

Corticosteroids appear to have markedly improved the outlook for patients with moderate to severe attacks of ulcerative colitis. There is still debate among physicians whether ACTH or corticosteroids provide the most benefit. My personal regimen is to start the patient on intravenous hydrocortisone (100 mg. by IV infusion, q.i.d.) and this appears to be a satisfactory regimen.

Topical corticosteroid therapy is also useful. Rectal drips of hydrocortisone (100 mg. and 120 cc. saline, b.i.d.) are beneficial but should be combined with systemic therapy. Azulfidine® is given as with a moderate attack of ulcerative colitis.

Antibiotics are commonly used in the severely ill patient with ulcerative colitis. The rationale behind using antibiotics in patients other than those with toxic dilatatation is not convincing but it is difficult to argue against their use. My usual preference is not to use antibiotics except in patients with toxic dilatatation or massive bleeding.

Psychological support is very important for these critically ill patients. Formal psychiatric evaluation is usually not appropriate. The patient should be placed in the best possible hospital surroundings and not placed in an open ward or in an intensive care unit. Psychological support from the patient's physician and nursing personnel is very important in determining the eventual outcome of the attack.

TOXIC DILATATION OF THE COLON

This dreaded complication of severe ulcerative colitis can also be seen in diseases of the colon such as Crohn's disease, amebiasis and others. It is, however, far more common in diffuse ulcerative colitis. It is recognized on a flat plate of the abdomen by the presence of dilatation particularly in the transverse colon. The normal gas filled colon may be as wide as 6 cm. in the area over the lumbar spine but should not exceed this in normal situations. In addition to the loss of normal haustrations, abnormalities of the gas shadow such as polypoid projections into the lumen of the bowel are usually seen and these aid in distinguishing toxic dilatation from a normal gas filled colon. Toxic dilatation of the colon is certainly one of the most difficult medical emergencies to manage. The decisions required during the course of therapy will tax the clinical judgement of even the most experienced physicians.

From the time the patient is admitted to the hospital with severe or moderately severe ulcerative colitis, the physician should be in consultation with a general surgeon. If both the internist and surgeon are able to follow the course of the illness together, a more intelligent decision as to timing of colectomy, if necessary, can be made.

The physician should take a careful look at possible precipitating factors which may have contributed to the onset of megacolon. Some are treatable, many are preventible. Perhaps the most common precipitating factor appears to be the barium enema. As mentioned before, this should probably be avoided during an acute attack. Other factors such as hypokalemia, hypoalbuminemia, anticholinergics and opiates should be searched for and eliminated. Anticholinergics have very little place in the therapy of ulcerative colitis, particularly in the severely ill patient.

Management of toxic dilatation of the colon is similar but more intensive than management of a more severe attack of colitis without megacolon. It includes: 1.) Nasogastric suction 2.) Treatment of fluid, electrolyte and colloid abnormalities 3.) Correction of anemia 4.) Systemic corticosteroids 5.) Systemic broad spectrum antibiotics (clindamycin or lincomycin should not be included) 6.) Rectal steroids 7.) Azulfidine® .

The patient must be carefully observed with the realization that while he is on high doses of steroids his apparent well being may not correspond to the true clinical situation. It is important to obtain a flat and upright film of the abdomen at least every 24 hours and one should look for intramural or free peritoneal air which would indicate impending or actual perforation of the bowel. After good medical management has been established, any further increase in the diameter of the colon or uncontrolled bleeding should be taken as an indication for surgical intervention. If the patient does not improve significantly within 48-72 hours after initiation of a medical regimen, this would be another indication for surgery. The trend in recent years has been toward earlier surgical intervention and this does appear to have improved the overall mortality of toxic megacolon.[3,4,5,6]

The choice of operations at most hospitals is ileostomy and total proctolectomy. However, because these patients are frequently poor surgical risks, excision of the rectum will often need to be deferred to some later time.

The physician treating ulcerative colitis must always remember that he is dealing with a curable condition. Although ilesotomy and panproctocolectomy seem awesome to both patient and physician, once performed, the patient rarely regrets the decision. The physician should do his best to avoid the life threatening consequences of severe, acute colitis through preventive means and vigorous treatment early in the attack.

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Obstetrics and Gynecology

Sodium In Pregnancy Part I

Harold Kaminetzky, M.D., FACOG

Donald J. Ziehm, M.D., Editor

The sodium ion and its relationship to fluid retention in pregnancy has become an important subject. Unfortunately, few clinicians take time to analyze the reasons for their own particular clinical routine when dealing with the edematous gravida. Why should (as classical teaching has dictated) pregnant women limit salt intake. Numerous reasons for this tendency have been advanced: 1) pregnant women often become edematous; sodium is the key ion in water balance, extra- and intracellular fluid; therefore somehow during pregnancy pregnant women fail to manage them properly; 2) additional substantiation was presented by Dieckman who demonstrated an aggravation of the symptoms in toxemic women following saline infusion; 3) non-pregnant patients who ingest large amounts of salt eventually tend to develop essential hypertension in greater numbers than people with a normal salt intake; 4) in the treatment of chronic essential hypertension blood pressure decreases significantly following reduction of dietary sodium. Consequently a clinical dogma developed that, if pregnant women would restrict salt intake, prophylaxis against the development of toxemia would result. However, it is now known that the normal pregnant woman actually has difficulty retaining all of the sodium necessary to carry out the normal physiology of pregnancy. There is no evidence to indicate that abnormal handling of sodium may produce toxemia.

What is the support for this conclusion? Pregnancy mandates an increase in the size of the maternal intercellular and intravascular fluid compartments. Of the 6-8 liters of water stored by the average gravida, 4-6 liters are intercellular. Furthermore, maternal tissues increase: about 3 pounds of weight are added in breast tissue, about 3 pounds in the enlarged uterus, approximately 7½ pounds of fat, 1½ pounds of amniotic fluid, and 1 pound of placenta. Approximately 60% is water, which is true of all tissue. The pregnant woman must retain about 19,500 mg. of sodium to produce this amount of water. A superb mechanism is present for accomplishing this during pregnancy. Chorionic gonadotrophin is secreted following fertilization, necessary to support the function of the corpus luteum (the production of estrogen and progesterone) which persists until placental estrogen and progesterone are elaborated.

Progesterone is natruretic and probably exerts its effect in two ways: 1) tubular reabsorption of sodium is diminished; 2) arteriolar tone is decreased which is caused by the baroreceptors in the kidney. The stimulus for natruresis is thus established. The progesterone-induced arteriolar vasodilation produces hypovolemia, while decreased tubular reabsorption of sodium causes hyponatremia. These changes are detected by the juxta-glomerular apparatus in the kidney and renin is secreted. Renin, reacting with renin substrate, is converted to Angiotensin I. Lung converting enzyme then acts on Angiotensin I to form Angiotensin II (a potent vasoconstrictor except during pregnancy). Angiotensin II stimulates the adrenal to secrete aldosterone (producing increased tubular reabsorption of sodium) and the neurohypophysis to secrete ADH (which causes water retention). This is the normal mechanism during pregnancy.

In the normal non-pregnant woman the average glomerular filtration rate is 120 ml/min., and the average circulating serum sodium 140 meq/L. Pregnancy causes

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an increase in the GFR by about 50%. Approximately 24,000 mg. of sodium will be placed into the glomerular filtrate per day in the non-pregnant woman. It is obvious that a very efficient mechanism for recall of this sodium must exist or the individual would soon die of circulatory collapse. Because of the 50% increase in the GFR during pregnancy, approximately 36,000 mg. of sodium are filtered daily. Without reabsorption of this additional sodium the gravida would certainly collapse. The kidney is one of the few organs that filters critically necessary substances (sodium and glucose) and also has means for reabsorbing them. How is this accomplished during pregnancy? The rationale for increase in aldosterone secretion becomes obvious. Since the amount of sodium presented to the macula densa is greater during pregnancy, additional renin is secreted. Angiotensin I and II production is enhanced. The outcome is increased reabsorption of sodium. Consequently, the average pregnant woman is in superb balance. For example, of the 22 pounds of tissue gained during pregnancy, 60% is water. To provide this amount of water 19,500 mg. of additional sodium must be retained. GFR in the non-pregnant patient is 120 ml/min., plasma sodium is 140 meq/L.

The quantity of sodium filtered each day is about 24,000 mg. Because of the 50% increase in GFR during normal pregnancy, 36,000 mg. of sodium are filtered. Assuming the normal gravida ingests 200 mg. of sodium daily, only 35,800 mg. of sodium will need to be reabsorbed through the renal tubules. If all else remained equal, but tubular reabsorption increased by only 0.1%, then 36 mg. of sodium would be added to the body stores. The amount of water required to maintain isotonicity in the extracellular fluid would give rise to a daily weight gain of 0.5 pound. Therefore, during pregnancy, 36,000 mg. of sodium are put into the glomerular filtrate, and all must be reabsorbed. The kidney must be a very efficient organ in handling sodium transfer during pregnancy since, if only miniscule amounts of sodium are retained in excess of the total put into the glomerular filtrate, plus dietary intake, 0.5 pound of weight must be added in order to insure isotonicity of extracellular fluid. Because of all of these normal mechanisms the average pregnant woman has little difficulty balancing sodium with her water intake and the need to retain sodium for her tissue needs, the tissue needs of the developing fetus, and the changes in the intravascular and intracellular compartments.

What happens to aldosterone secretion during toxemia of pregnancy? It was previously thought that generalized edema developed in toxemia because of abnormal sodium retention. In reality, in genuine toxemia or pre-eclampsia, aldosterone secretion is less than that in the normal pregnant woman. Blood volume and extracellular fluid are both decreased or contracted. That the fundamental problem in pre-eclampsia is abnormal handling or retention of sodium has not been documented. (*to be continued*)

Oncology

Malignant Melanoma: New Clinical Research Directions At Arizona Health Sciences Center

Frank L. Meyskens, Jr., M.D.

Editors:
Stephen E. Jones, M.D.
Robert H. Thoeny, M.D.

Progress in the treatment of malignant melanoma has reached a plateau.[1,2] The immunostimulant BCG probably has a place in the treatment of resected, poor-prognosis (mucosal; Stage I Clark level 3 to 5; and Stage II) malignant melanoma;[3-5] however, its definite role in preventing relapse and increasing survival is still unclear. The treatment of metastatic melanoma has not significantly improved since the introduction of DTIC,[6] although the combination of BCNU, hydroxyurea, and DTIC may have increased efficacy.

At the University of Arizona Health Sciences Center (AHSC) two new approaches to the management of this aggressive malignancy have been recently initiated: (1) predictive *in vitro/in vivo* correlation of selected drugs in metastatic melanoma and (2) the addition of an immunopotentiator (high-dose Vitamin A) to the immunostimulant BCG for the adjuvant treatment of resected, poor-prognosis melanoma.

IN VITRO/IN VIVO DRUG SENSITIVITY CORRELATION STUDY OF METASTATIC MALIGNANT MELANOMA

The development of an *in vitro* colony-forming assay for stem cells from certain human tumors was recently discussed by Sydney Salmon in this column.[7] A

*Editor's note: Any physician interested in discussing this further or referring patients suitable for these two Arizona studies can contact either Dr. Meyskens (882-7228) or the main Hematology-Oncology Office (882-6372 or 882-6373).

From: Section of Hematology and Oncology, Department of Internal Medicine, University of Arizona Health Sciences Center, 1501 North Campbell Avenue, Tucson, AZ 85724.

TABLE 1

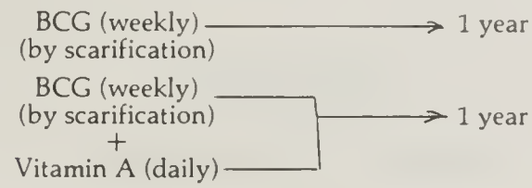
Melanoma in vivo/in vitro drug sensitivity correlation study basic design

Patients Needed	Patients with *metastatic melanoma* and lesions which can be biopsied easily (e.g., skin, nodes)
Study Design	
In vitro:	drug sensitivity for DTIC, BCNU, Actinomycin D
In vivo:	DTIC therapy as soon as biopsy taken for a minimum of 2 courses. At the time of relapse patient will receive either BCNU or Actinomycin D depending on the *in vitro* drug sensitivity results. If a relapsing patient is sensitive to neither, he will be rebiopsied and restudied with a different set of drugs.

TABLE 2

Randomized study of BCG versus BCG and Vitamin A for resected, poor prognosis melanoma

Patients Needed	Patients with mucosal (any site), Stage I (level 3 to 5), or Stage II melanoma.
Study Design	A randomized study with stratification for age, sex, stage.



quantitative measure of tumor stem cell sensitivity to anticancer drugs was developed in this system. However, what is not clearly known is whether the positive and negative sensitivities obtained *in vitro* will predict the sensitivity of the patient's tumor *in vivo*. To successfully test this *in vitro* system for *in vivo* (clinical) relevance in a reasonable amount of time, several conditions are required: (1) the tumor should be one that grows with high frequency in the target media; (2) the tumor should be one that is frequently accessible for biopsy; (3) there should be good patient accrual; (4) the tumor should be one for which the therapeutic choices are limited and poor; (5) a well-designed *in vitro/in vivo* study should be available; and (6) there should be excellent understanding and cooperation from the clinicians involved.

For reasons (1) to (3) we have chosen metastatic malignant melanoma as the tumor type. Eligible patients with accessible metastatic melanoma will have biopsy of their tumor and the establishment of an *in vitro* tumor stem cell culture which will be tested against three drugs commonly used against melanoma. Since it takes 2-3 weeks to learn the results of this test (similar to antibacterial antibiotic testing), all patients will be begun on single-agent treatment for melanoma with DTIC. If the patient has stable disease or manifests an objective response to DTIC, this agent will be continued. If tumor progression occurs, the choice of the next drug to be used will be based on the results of the *in vitro* assay. In this way, we can verify in a small number of patients whether the laboratory test predicts clinical response. The type of patients needed and the study design are outlined in Table 1.*

BCG AND HIGH-DOSE VITAMIN A FOR RESECTED, POOR-PROGNOSIS MELANOMA

Considerable data from animal studies suggest that high-dose Vitamin A is effective as an immunostimulant when the tumor burden is low ($\sim 10^5$ cells) and as an immunopotentiator when the tumor burden is increased ($\sim 10^7$ cells), especially in animal melanoma.[8,9] The relapse rate from resected mucosal, Stage I (Clark level 3-5), and Stage II melanomas is high and 5-year survival poor.[4] The immunostimulant BCG may have efficacy in preventing or delaying recurrence of resected malignant melanoma.[3,4] Over the past 3 years it has been our policy to administer adjuvant BCG only to high risk patients after adequate surgical therapy.[1] The present study is designed to learn whether the addition of Vitamin A to BCG offers these patients greater benefit than BCG alone. The type of patients needed and the study design are outlined in Table 2.*

SUMMARY

The successful treatment of malignant melanoma has largely reached a plateau. New approaches to this aggressive and elusive malignancy are sorely needed. We describe two such approaches in this report: (1) the *in vitro* prediction of *in vivo* response in metastatic melanoma and (2) the addition of high-dose Vitamin A to BCG for resected, poor-prognosis lesions. Close cooperation and referral of suitable patients from Arizona physicians have contributed substantially to the success of such clinical studies in the past. Patient categories that are needed for successful completion of these studies in patients with melanoma are outlined in Tables 1 and 2.

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Epiphora

Or the "Tearing Eye"

Robert Rubenzik, M.D.

"Doctor, my eye keeps running tears. What can be done about it?" This is a common complaint, often arising in a busy practice. This article will discuss some aspects of epiphora, or tearing, from a practical standpoint.

When it comes to tearing, there are two factors to consider—tear overproduction and tear duct obstruction.

Tears are naturally secreted by glands in the conjunctiva—the so called accessory lacrimal glands—at a very low rate (approximately 1cc/day). When the cornea becomes dry or irritated a reflex is initiated which causes the main lacrimal gland to secrete copiously. The gland may also be stimulated centrally, as in crying. When the main lacrimal gland secretes, epiphora nearly always results, as the drainage system is not capable of handling such a fluid overload.

The test usually used to assess tear production consists of placing a standardized strip of filter paper in the eye and measuring the amount of wetting seen in a specified time interval (Schirmer test). If tear production is *high*, reasons for hypersecretion must be sought, usually related to corneal irritation. This is often due to entropion with the lashes abrading the cornea. Sometimes if tear production is *very low*, as in Sjogren's syndrome, the cornea may dry out and stimulate intermittent reflex secretion from the main lacrimal gland.

Careful examination of the cornea with the slit lamp yields more relevant information about the presence or absence of corneal irritation than any other test.

When the cornea looks normal and when the Schirmer tests reveals a normal rate of tear production, tear duct obstruction must be suspected. There is nothing mysterious about a plugged-up tear duct. Partial obstructions, however, require practical judgement. If tearing occurs very infrequently, surgery may not be worth the trouble, risk, and expense to the patient. A completely obstructed tear duct causes no symptoms in some people, while in others tearing is constant and troublesome. Infrequently, a person with an obstructed nasolacrimal duct will develop an abcess in the tear sac, necessitating prompt drainage.

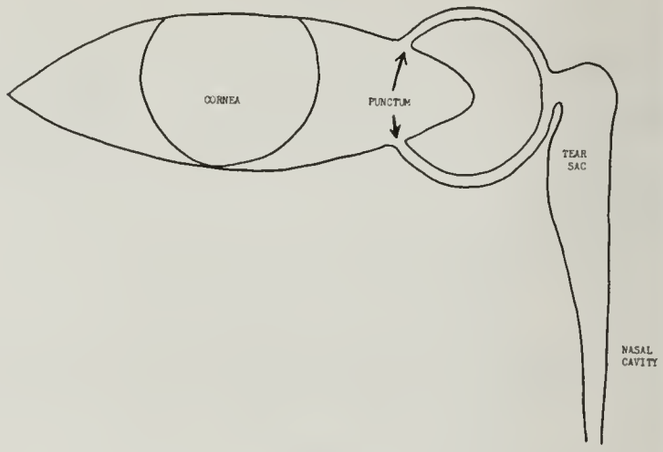


FIGURE 1

A diagram of the lacrimal system. In order for tears to find their way into the system, the lower punctum is of greater functional significance than the upper. Ectropion in the medial aspect of the lower lid prevents tears from gaining access to the drainage channels.

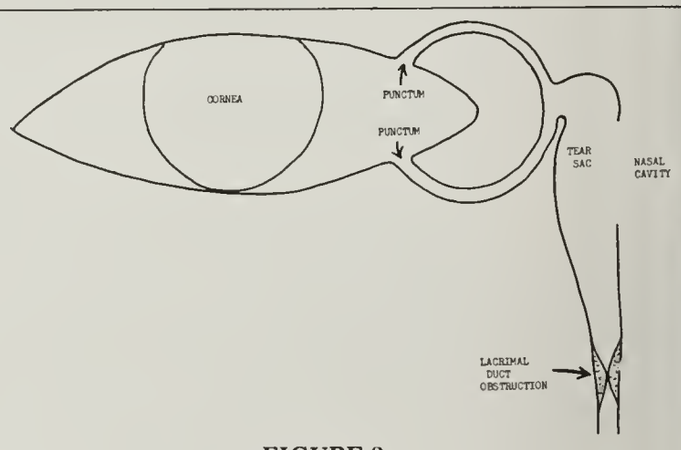


FIGURE 2

In this case the nasolacrimal duct is totally obstructed. The obstruction is bypassed by surgically anastomosing the tear sac to the nasal cavity [dacryocystorhinostomy]. This may be done under local or general anesthesia, and yields a high percentage of success.

When a patient is found to have insufficient drainage of tears, the next thing to examine is the eyelid. The position of the punctum (the external opening of the tear duct in the eyelid) must be evaluated relative to the eyeball. If the patient has an ectropion, the tears cannot find their way into the ductwork. One simple rule: The punctum in the lower lid is normally opposed to the globe, and cannot be seen from the front. If the opening is visible from the front of the patient, the punctum is malpositioned (Fig. 1).

Irrigation of the lacrimal system will reveal the degree, and often the location, of the obstruction. The system may be narrowed at any point. Treatment consists of enlarging or bypassing the obstructed area.

From 421 N. 18th St. Ste. 105, Phoenix, AZ 85006.

If a probe may be inserted through the tight area, the system may be intubated temporarily with silicone rubber tubing. Otherwise, surgical repair is required, the most common procedure being the dacryocystorhinostomy, which anastomoses the tear sac with the nasal cavity (Fig. 2).

All in all, there is probably nothing wrong with giving the patient a trial of eyedrops, i.e. something to "Get the red out." If conservative measures fail to bring relief, a systematic evaluation by an ophthalmologist is in order. The treatment must be directed toward the specific site of irritation or obstruction in order to produce a cure.

Radiology

Case of the Month #30

Frederic C. Park, M.D.
John C. Bjelland, M.D.
Bert Sosnow, M.D.



FIGURE I
Plain film of the abdomen. Note multiple bony densities in the pelvis [arrows].



FIGURE II:
Supine film of the abdomen from small bowel examination. Note large mass displacing small bowel loops.



FIGURE III:
Midline longitudinal sonogram delineates large abdominal mass.

An alarmed twenty year old female sought immediate medical attention after she became aware of a "grapefruit-sized" mass in her abdomen. In retrospect, the patient admitted she had noted a gradual increase in abdominal girth during the past year. She thought this might be due to maintaining a normal daily caloric intake, while remaining relatively physically inactive, after she had undergone a partial transverse colectomy eighteen months earlier. Her doctor had assured her the final surgical pathologic report indicated her polyps were of the benign, adenomatous variety.

On physical examination, prominent prognathia and a "cherub" facies were noted and a large, firm, non-fluctuant, non-movable, mid-epigastric mass was palpated.

The above diagnostic medial imaging studies were obtained (see *Figures* I-III and the respective legends). What is the diagnosis?

From: Radiology section, U of A Health Sciences Center, Tucson, AZ 85724.

Gardner's Syndrome: Extra-Colonic Manifestations



FIGURE IV:
Examination of the skull demonstrates mandibular disfigurement due to osteomata.

Gardner's syndrome is an inheritable Mendelian dominant disorder consisting of the following clinical triad: polyposis coli, osteomata (multiple osteomas), and various cutaneous and/or soft tissue tumors. This case is representative of the two primary extra-colonic manifestations.

The benign adenomatous colinic polyps have a known propensity to undergo malignant transformation.[1] This usually occurs about fifteen (15) years after the onset of polyposis.[2] If this component of the symptoms and signs complex remains undetected, or untreated, most patients will either develop an advanced neoplasm or die from metastatic disease prior to the age of fifty (50).[3] Therefore, the earliest clinical recognition and identification of the syndrome is essential for the most optimum delivery of health care and genetic counseling. Fortunately, the average Gardner's patient initially presents prior to the age of twenty (20) with rectal bleeding secondary to polyposis, which leads to the early diagnosis and proper treatment plan.

However, the second most frequent clinical presentation is due to the soft tissue component of the syndrome. Often the patient initially seeks the clinician for evaluation of the following internal or external soft tissue lesions: fibromas, epidermoid cysts, desmoid tumors, or post-traumatic/surgical keloid formation. These disorders raise the clinical suspicion for Gardner's syndrome. Patient referral for detection and evaluation of the radiological aspects of the disorder (*i.e.*, osteomata, fibromata, and polyposis) can then be made to confirm the tentative diagnosis.

This twenty year old female had previously presented with hematochezia due to polyposis and had undergone a partial transverse colectomy. Post-operatively the patient developed the incidious onset of increasing abdominal girth due to the slow growing mid-epigastric mass demonstrated in *Figures* II-III. *Figure* I shows multiple areas of osteosclerosis in the pelvis. Diagnostic possibilities for these densities include: (1) a blastic neoplastic process, (2) multiple bone islands, (3) multiple bone infarcts, and, (4) osteomata.

The radiographic character of these densities, the patient's age group, and the previous surgical history should suggest a tentative diagnosis of Gardner's syndrome rather than metastatic disease. Osteomata are present in over 50% of the patients with this hereditary disorder. Such characteristic large osteomata *alone*, particularly in the skull or mandible (see *Figure* IV), strongly suggests Gardner's syndrome. Occasionally, other oral pathologies (*e.g.*, multiple unerupted teeth, dentigerous cysts, and odontomas) are also present as diagnostic adjuncts accompanying the syndrome.[4]

The most disturbing facet of this case is establishing the proper etiology of the abdominal mass. The sonogram demonstrated a large, non-cystic abdominal mass. The absence of mural or mucosal radiographic abnormalities in the adjacent, extrinsically displaced small bowel loops suggests this surgical lesion is benign. A large fibromatous connective tissue mass (having the appearance of a gigantic keloid) was exised from the root of the mesentary at the site of the previous transverse colectomy. Such mesenteric fibromatosis is known to occur following colectomy in Gardner's patients and this is a common postoperative complication.[5]

While Gardner's polyposis results in adenomatous proliferation, Peutz-Jegher's syndrome is characterized by the presence of hamartomas of the colonic mucosa. However, Peutz-Jegher's usually presents with small bowel involvement, often as an intussusception. Other rare polyposis syndromes include Turcot's, with adenomatous colonic polyps associated with CNS tumors and Canada-Cronkite syndrome in which the adenomatous polyps are associated with etodermal dysplasia.[6]

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Adverse reactions to theophylline are related to serum levels and are usually not a problem at concentrations below 20 mcg/ml. Of 45 patients studied[1] after a single dose, only seven reported adverse reactions. The most common reaction was a feeling of lightheadedness by three of these seven patients.

Reference 1 Data on file, Mead Johnson Pharmaceutical Division

Indications: For the symptomatic treatment of bronchospastic conditions such as bronchial asthma, asthmatic bronchitis, chronic bronchitis, and pulmonary emphysema.

Dosage: Quibron—Adults: 1-2 capsules or 1-2 tablespoonfuls elixir every 6-8 hours. Children under 12: 4-6 mg theophylline/kg body weight every 6-8 hours.

Quibron-300— Adults: 1 capsule every 6-8 hours.

Theophylline dosage may be cautiously increased to 2000 mg/24 hour in adults and 9 or 10 mg/kg every 6 hours in children. Monitoring of serum theophylline levels at higher dosages is recommended.

Precautions: Do not administer more frequently than every 6 hours, or within 12 hours after rectal dose of any preparation containing theophylline or aminophylline. Do not give other xanthine derivatives concurrently. Use in case of pregnancy only when clearly needed.

Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea, and vomiting. The frequency of adverse reactions is related to the serum theophylline level and is not usually a problem at serum theophylline levels below 20 μg/ml.

How Supplied: Quibron Elixir: Bottles of 1 pint and 1 gallon. Quibron Capsules: Bottles of 100 and 1000 and unit-dose packs of 100. Quibron-300 Capsules: Bottles of 100.

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Rheumatic Diseases

Histocompatibility Antigens And Rheumatic Diseases

Benjamin K. Harris, M.D.

Histocompatibility antigens (HLA) are glycoproteins on the surface of nucleated mammalian cells; they are detected by lymphocyte cytotoxicity tests using monospecific antisera. In 1973, investigators from Los Angeles and London[1,2] reported that 90% of patients with ankylosing spondylitis were positive for HLA-B27, whereas the frequency of this antigen in control populations was 7-8%. This was the strongest association ever uncovered between an HLA haplotype and a specific human disease.

Subsequently, HLA testing was vigorously pursued in a group of inflammatory rheumatic diseases that are usually negative for rheumatoid factor and often culminate in spondylitis, the so-called rheumatoid variants. In Reiter's syndrome, with or without spondylitis, more than 90% of patients were positive for B27,[3] and in psoriasis with spondylitis and inflammatory bowel disease with spondylitis the frequency of B27 was about 70%.

Exactly how the presence of HLA-B27 relates to the development of ankylosing spondylitis or the other rheumatoid variants is poorly understood. It is possible that the gene for B27 may be closely linked on the C-6 chromosome to immune response genes which, when confronted by certain environmental factors, lead to the induction of specific rheumatic diseases. Some of the environmental factors have been identified in Reiter's syndrome where the full clinical picture has emerged following Shigella or Salmonella infections in B27 positive patients.

Testing for histocompatibility antigens is a costly, time-consuming process, and supplies of monospecific antisera are limited. There are few guidelines for the clinician hoping to use HLA testing efficiently in the differential diagnosis of connective tissue disorders. Our experience with histocompatibility antigens in rheumatic diseases during the past few years suggests that B27 testing is helpful in evaluating the following clinical problems:

1) The young male with persistent back pain suggesting an inflammatory origin who may have equivocal roentgenographic findings, 2) the patient with an incomplete picture of Reiter's syndrome;[4] in this situation, a more precise diagnosis gives useful prognostic information in that most patients with Reiter's syndrome do *not* develop chronic arthritis, and 3) the patient with either psoriasis or inflammatory bowel disease and peripheral arthritis; here, positivity for B27 identifies those patients at greatest

From: Arthritis Associates, 1010 E. McDowell Road, Phoenix, AZ 85006.

risk for development of spondylitis.

With additional time and study, it is likely that large scale screening for B27 positive individuals will be warranted. Calin and Fries[5] have shown, in a study of apparently healthy blood donors, that 20% of B27 positive subjects may eventually develop ankylosing spondylitis. Extrapolating from their data, it is possible that 1.4% of the United States population (7% of controls are positive for B27 and 20% may have disease expression) or more than three million persons may have ankylosing spondylitis. However, in the process of detecting those individuals susceptible to spondylitis, it should be realized that 80% of those positive for B27 will not develop rheumatic disease, and the testing will fail to pick up the 10% of spondylitis patients who are B27 negative. Of the 20% of B27 positive patients who later develop spondylitis, it is assumed that early diagnosis will have a favorable impact on the natural history of the disease. We in clinical rheumatology believe that early diagnosis with prompt institution of physical therapy and nonsteroidal anti-inflammatory agents correlates with less subsequent deformity, but solid data to support this assumption are lacking.

Additional caution is needed in the interpretation of B27 data in view of the fact that 7% of a control population will be B27 positive. We have recently seen one patient with a history of Reiter's syndrome who was B27 positive and who subsequently developed rheumatoid arthritis,[6] and another with B27 positive ankylosing spondylitis who, many years later, presented with polymyalgia rheumatica. There have been rare reports of patients who have features of both ankylosing spondylitis and rheumatoid arthritis. At the present time, there is no reason to believe that positivity for B27 confers any protection against unrelated rheumatic diseases.

The availability of new diagnostic tests always poses a dilemma for the conscientious physician who wishes to avail his patients of the latest advances without subjecting them to soaring laboratory fees. It is reassuring, in this regard, to note that Calin and Fries[7] have found a careful clinical history to be as sensitive and more specific as a screening test for ankylosing spondylitis than checking for HLA-B27.

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Medicine In Mexico

VIII. Urban Health Care — Mexico City

Charles C. Daschbach
Ramon Hugo Martinez Lozano

The topic of urban health care in Mexico today is intimately linked with a concern about the future of her largest metropolitan area, Mexico City. Known as the Federal District, Mexico City is currently the third largest urban complex in the world with over 9 million inhabitants. She has the highest annual growth rate among major cities (5.4%) and by the year 1994, U.N. demographers predict that she will become the most highly populated area on earth. She will have a population of over 31.5 million persons by the turn of the century, increasing her population density to over 21,000 persons/km^2. This trend is due to one of the world's highest population growth rates (3.5%) and a rural-urban migration estimated at 1,000 persons per day. Within the last year, preparations have begun to cope with the future stress that this population will place on health care delivery and public health standards.

A current profile of Mexico City shows 24% of the nation's population living inside the city limits, which covers an area of 1,499 km^2 or 0.5% of Mexico's total land surface. (This figure does not cover persons who live in the Valley of Mexico outside the Federal District.) In 1976 there were 1.6 million family dwellings, 28% with only one room, 25% with two rooms and 17% with three rooms. Seventy percent of the homes had indoor running water and 78% had adequate drainage. One government study estimated that 20%

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of the families had problems associated with the break-up of the family unit. The most depressed barrios in the valley are probably the municipalities of Netzahualcoyotl and Tlalnepantla which lie outside the city limits in the State of Mexico. Current government efforts to aid their large (1.5 million) population are using the Mexican rural modular-pyramid approach, which employs central coordinating hospitals, clinics (*Centros de Salud*), and health education sub-clinics (*Casas de Salud*).

In the Federal District proper, 250 hospital units provide 26,000 beds or 2.8 beds per 1,000 inhabitants. Of these, 67% are provided by government socialized health programs (IMSS, ISSSTE and SSA-DF) with the rest belonging to private independent institutions. Over 3.6 million city inhabitants are covered by government sponsored health programs, covering on-the-job injuries, sickness, maternal care, invalidity, old age and death expenses.

In 1976 there were 19,000 physicians (35% of the national total) practicing in Mexico City, with one doctor per 475 inhabitants compared to one per 1250 for the rest of the country. Of this number, almost 78% receive a salary in the public sector and 22% or 4,200 doctors are in private practice only. The city has 12,500 qualified nurses (one per 720 pop.), 16,000 nursing auxiliaries (one per 563 pop.), and 3,200 dentists (one per 2,813 pop.). This shortage of non-physician health providers is the most serious future manpower problem faced by Mexico City.

A major step in the preparation of the Federal District's future was the recent formation of the Joint Urban Commission comprised of government officials from the city and the adjacent states in the valley of Mexico. Future plans call for an Autonomous Central Mexico Urban Complex which will spill into five states with an estimated population of 40 million persons. An integral part of this master plan is the anticipation of this population's health needs—drinking water, sewage, communicable disease control, chronic and acute medical care. The public health challenge will be the greatest in the history of urban man.

Disaster Prevention

Planning for a Rock Concert

John H. Hughes, M.D.

The field of Emergency Medicine has included in its areas of concern the management and prevention of disasters.[1] Rock festivals have led to illness and injuries greater than could easily be accommodated by health resources in multiple communities in the past. Accordingly, when the University of Arizona agreed to host a rock concert in its football stadium as a benefit for the Arizona Chapter of the American Heart Association, the health planning was approached as a potential disaster. Rooen[2] had noted 8 points for organization of health services for such events. Based on experiences in Canada prior to 1971, he suggested that the health facility receiving patients from such an event should plan for 1 casualty per 500 paid attendance per hour of concert. Based on those figures, a 6 hour concert in a stadium of 50,000 people would be expected to generate 600 casualties.

Preliminary Planning

In order to minimize illness and injury, a meeting was held one month prior to the event which included the President and staff members of the Heart Association, the Director of Emergency Services for the University of Arizona, other physicians and nurses in emergency care volunteering their services, a representative of the rescue unit of the Fire Department, representatives of each private ambulance company providing paramedical care to the Tucson area and representatives of the local unit of the Army National Guard. It was mutually agreed that no alcoholic beverages or obvious drugs be allowed into the stadium. It was also agreed that the pattern of medical care provided within the stadium would be transportation and triage. Any individual found significantly ill or injured was to be taken to appropriate sites for evacuation by vehicles manned by Paramedics to the nearest hospital. Since the University Hospital was the closest health facility, the Heart Association staff requested that it serve as the receiving Hospital. Because of the predicted heat, special arrangements were made for generous water supply within the stadium. First aid stations were adequately marked and telephones at each station were in proper working order. It was agreed that two squads of four men each serving as stretcher bearers would be present on each side of the field wearing luminescent markers over their shirts.

A cooperative plan between campus police and the city police, county sheriff's deputies, and the Arizona Department of Public Safety were developed. Beyond this, a group of 300 students volunteering to assist in crowd control were recruited. Health personnel, law enforcement personnel and maintenance workers used portable communication handsets working on separate frequencies. All communications with a ground control station were by standard telephone to a restricted number.

Two weeks prior to the event the Director of Emergency Services provided to the University Administration a consultation on physical hazards and possible problems which might be corrected prior to the event. The plans for the event were presented formally to the Disaster Committee of University Hospital and it was agreed that this be considered an official Disaster Drill. Appropriate contingency plans were developed for both utilizing part of the Hospital Disaster Plan and mobilizing appropriate extra staff. Arrangements had already been made to transport any overflow to an alternate hospital in the community. Beyond this, an organized disaster plan exists within the community for which other hospitals could easily be utilized.

On the morning of the event, the travel route for the Paramedical vehicles was checked and secured by law enforcement. Crowds of individuals arriving early were surveyed in order to predict the types of drugs and injuries which might be expected. Four hours prior to the opening of the gates, some 20,000 people were in line consuming mainly beer and hot sunshine. A cooperative attitude clearly existed at that time between all law enforcement and health personnel as well as the students helping to arrange the affair. This information was provided to the Triage Officer in the Emergency Room at University Hospital.

The Event

Over 67,000 people attended this event. The restriction on alcoholic beverages or any closed containers being brought into the stadium was strictly enforced. Accordingly, at the time the concert began the outside gates were overflowing with bottles, cans, and rubbish. This constituted a significant hazard to safety and was cleared as rapidly as possible.

The Casualties

In the total time prior to, during, and following the event, only 16 patients needed to be seen at University Hospital. One was sent directly to the Veterans Administration Hospital.

These included the following:

From: Dept. of Emergency Services, Arizona Health Sciences Center, Tucson, AZ 85724.

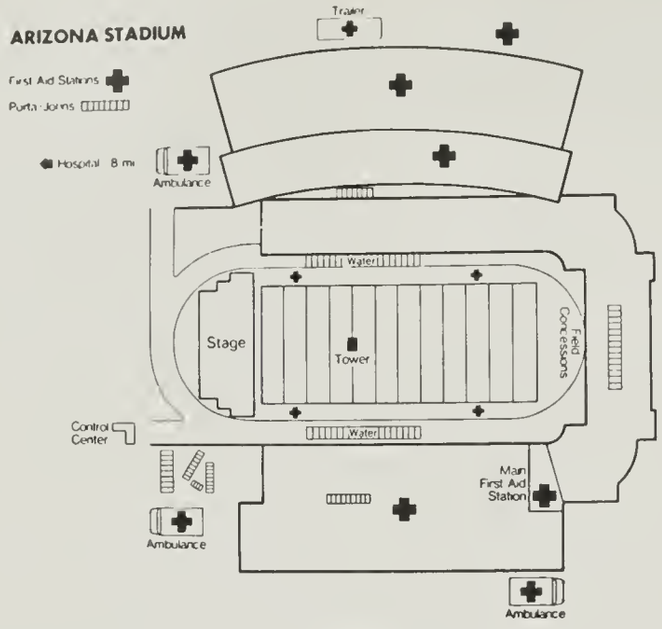


FIGURE 1

The immediate availability of health resources was felt to help minimize illness and injury.

1. An 18 year old male combative and confused, admitted for drug overdose. Laboratory determinations were positive for alcohol, phenobarbital and phenylpropanolamine.
2. A 22 year old male believed to have taken an overdose of phencycladine was treated and left the hospital two hours after arrival.
3. A 24 year old white female who had been fasting for 11 days who had three syncopal episodes, was treated and discharged.
4. A 28 year old white female whose blood ethanal level was 0.315 who had been treated and referred to the University police for any further problems.
5. A 17 year old white male who had taken cocaine and consumed considerable beer and rum prior to entry into the stadium. He was treated and discharged.
6. A 23 year old white male treated for heat exhaustion.
7. A young male thought to have taken Barbituates, admitted for overdose.
8. A 32 year old female with syncopal episodes secondary to dehydration.
9. A 25 year old male law enforcement officer struck on the back of the head with a bottle. The officer had his helmet on at the time, was alert at the time of evaluation but was admitted for observation.
10. A 16 year old male who contused his leg climbing under his car to get his hat.
11. A 41 year old male law enforcement officer who, while riding a mini cart backwards, struck his head on a cement pole for which he had a small laceration.
12. An 18 year old female buttock laceration secondary to sitting on broken glass.
13. A 17 year old male slipped in a water puddle and sustained a Jones fracture of the left foot.
14. A 21 year old male struck his right leg against a steel spike producing a slight laceration.
15. A 21 year old female with a corneal abrasion secondary to a flying object.
16. A 16 year old female who sprained her elbow when she was climbing over a fence and another person fell on her.
17. A 32 year old male veteran who was quadriplegic developed dizziness and was admitted to the Veterans Administration Hospital for heat exhaustion.

It should be noted that the aid stations were equipped only with water, ice, salt tablets, and supplies to cleanse and dress minor cuts and abrasions. Any problem requiring more major care was triaged to the Hospital. A total of 299 patients presented minor problems in 332 patient visits (some patients were seen at multiple aid stations).

One week after the event, a meeting was held involving University of Arizona Administration, law enforcement, health, students, maintenance and concessions. It was agreed that many factors contributed to maintaining a relatively orderly event. These included:

1. Advance publicity by the news media regarding suggestions regarding traffic patterns, and available health facilities. Advance notice that alcohol would not be permitted in the stadium was also provided.
2. The support by the performers in announcement of requests that the audience respect the stadium property and assist in requesting aid if any health problem arose.
3. Minor aggrevations to the crowd were avoided. When gates opened, there was no unnecessary crowding in spite of the need to enforce the no alcohol regulations at the gate. Concessions were available with soft drinks and adequate ice supply during the hot late afternoon.
4. Students maintained an attitude of respect for the school and the performers such that potential aggitators in the crowd met unreceptive responses.
5. With random seating on the field, a light and sound mixer tower in front of the stage blocked vision for anyone sitting directly behind. This then provided an aisle badly needed for the stretcher teams in such a situation.

Problems which were recognized included difficulty recognizing locations of personnel communicating over hand carried mobile transmitters. Inadequate dim lighting within the crowd during the event was considered a hazard factor.

Discussion:

It has been suggested that a disaster by definition occurs with little or no warning.[4] The need to identify vulnerable populations and the importance of developing methods and logistics for assessing and feedback reporting have been stressed in disaster management.[4] There is certainly evidence that both overcrowding and noise lead to violent and destructive behavior.[5] Thus, a crowded stadium gathered for a rock concert on a hot evening presented circumstances clearly conducive to injury. While advanced planning probably contributed significantly to minimizing illness and injury, other factors may contribute. Pringle[5] has suggested the need for new experiences and the need for responsibility are important to the mental health and development of all individuals. Lack of constructive mechanisms to fulfill these needs are parts of the emotional roots of violence and vandalism in his opinion. Since this event provided not only a new experience but an opportunity for responsibility by students, its emotional impact was largely positive, occurring early in the school year before anxieties and frustrations associated with school activities could provide disruption, basically a healthy situation existed. Hott[6] describes group violence as a self-destructive act borne out of hopelessness - either real or imagined. The lack of such misdirected, uncontrolled behavior might suggest some feeling of hope among the participants.

Conclusions:

Although a rock concert in a crowded stadium on a hot evening provides circumstances in which aggressive misdirected activity could lead to significant casualties and illness, such circumstances were averted in one event. A cooperative attitude between various health and law enforce-

ment agencies, student groups, and participants along with adequate advance planning probably helped to avert significant problems. Other behavioral factors may have contributed to this result.

The pattern of drug overdosage, alcohol intoxication, and trauma were seen here as in previous rock concerts. The planning steps are described as a guideline to assist others in planning of such events.

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R.S.V.P.

Warren F. Gorman

When you've shared with him that most intimate feeling, if he should say, warmly, with his back toward the ceiling,

"Thank you,"

after a moment like the symphony's when they crash the Cymbals, his words are the clerk's over the counter at Gimbel's,

when you feel so warm, and inside so placid, is he being sweet, with a faint touch of acid?

Should you act like the baby, annointed with talcum, who wriggled the signal, meaning "You've walcum"?

If he's Hispanic, and to your child is the father, should you lower your lashes and then say, "de nada"?

Or if you feel a new, pleasant tension, it isn't the time to say, "Don't mention it."

Lay it to rest. The quid pro quo's best - just murmur, absent mindly, "Thank you, sir, kindly."

U of A Grand Rounds

Polyuric Syndromes

Ulrich F. Michael, M.D.

Polyuria is defined as a persistent increase in urine output. Many clinical conditions are accompanied or even characterized by increased urine volumes of varying degrees. They vary widely in their clinical importance. At one extreme, they may simply represent an inconvenience to the patient, who has to arise several times each night to urinate. At the other extreme, they cause life-threatening fluid losses. In the arid regions of this world, like southern Arizona, any impairment in urinary concentrating ability constitutes a severe handicap to any person whose access to fluid is for one or the other reason limited.

In order to understand polyuric states it is important to understand factors that influence urine output. The volume of urine excreted at any given time depends on a) the state of the body's water balance, b) the amount of solute presented to the kidney for excretion and c) the renal capacity for concentrating and diluting the urine.[1]

Countercurrent concentrating mechanism

While the morphology of the mammalian nephron with its U-shaped loop had been described well over a hundred years ago,[2] the functional importance of this structural arrangement was not recognized until 1942. Werner Kuhn, a physical

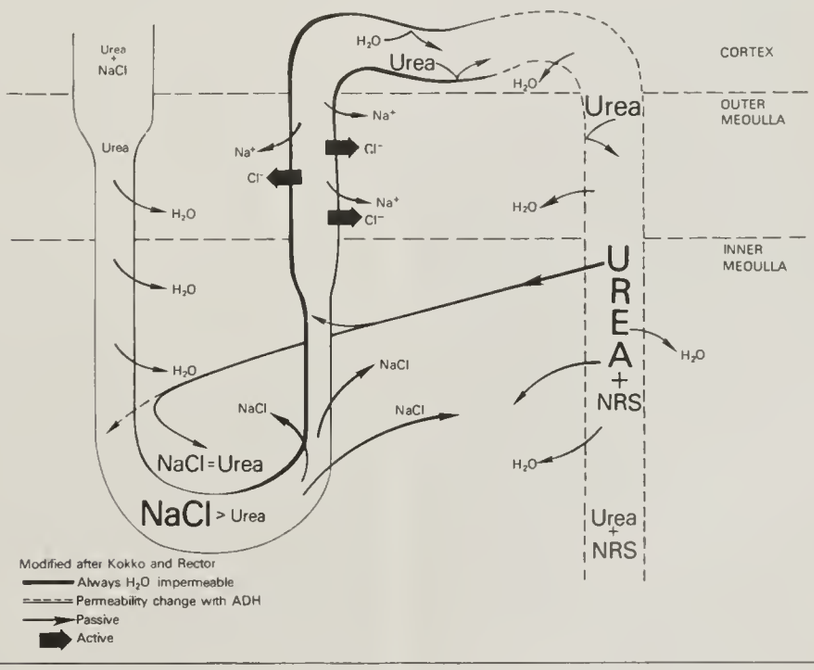


FIGURE 1.

Schematic drawing of a nephron unit. Concentrations are reflected by size and thickness of lettering. Note that NaCl concentration of tubular fluid is higher than that in interstitium at the bend of the loop and that urea concentration is highest in the early inner medullary collecting duct. NRS = non-reabsorbed solutes.

From: Veterans Administration Hospital, 3601 S. Sixth Ave., Tucson, AZ 85723. Dr. Michael, Assoc. Prof. of Medicine, Chief, Renal Section.

chemist, stressed the significance of tubular fluid flowing in opposite directions in adjacent nephron segments, a situation comparable to countercurrent heat exchangers, which allows the build up of a steep vertical temperature gradient with only a small horizontal gradient, which has to be generated with the expenditure of energy.[3] In order for a countercurrent concentration system to be operable in the kidney, Kuhn postulated three prerequisites: a) an energy source [Einzeleffekt (single effect)], b) countercurrent flow and c) differences in permeability in the tubular structures with opposing flows. There is agreement under renal physiologists that active salt transport out of the thick ascending limb of the loop of Henle provides the energy to drive the system. The ion actively reabsorbed is chloride, with sodium following passively.[4] The question which had puzzled students of the mammalian renal concentrating mechanism was: How does transfer of the energy from the outer medulla to the inner medulla and papilla occur? One solution would be an energy source in the inner medulla. Active salt transport by the thin ascending limb of the loop of Henle, a theoretical possibility, has purportedly been demonstrated in the golden hamster,[5] but in no other mammal has evidence of active salt transport by the thin ascending limb been obtained.[6] How then could the solute concentration in the inner medulla increase to a level which exceeds that of plasma in the cortex and the rest of the body by a factor of 4 in the human kidney and by a factor of 15-20 in some desert animals? Recently several models of the renal concentrating mechanism have been proposed, which are capable of explaining the accumulation of solute in the inner medulla/papilla region of the kidney without the prerequisite of active transport out of the thin ascending limb. These models are referred to as "passive" even though it should be clearly understood that concentration of urine requires energy, which is generated in the cortex and outer medulla and transferred to the inner medulla by means of urea recirculation (vide infra). In this brief outline I shall only describe the model proposed by Kokko and Rector,[7] based on studies of the transport and permeability characteristics in the isolated nephron of the rabbit (Figure 1, Table I). Let's begin with the energy source, which consists of active salt transport confined to the thick ascending limb of the loop of Henle (ALH). It has been clearly demonstrated that the ion actively transported in the thick ALH is chloride, while sodium follows passively out of the tubular lumen.[4,8] Furosemide or ethacrinic acid inhibit chloride transport at this site of the nephron.[9] Both diuretics are capable of dissipating the concentration gradient which has been built up between cortex and inner medulla in a matter of a few minutes.[6] Because water permeability is low in the thick ALH, active salt transport renders its content dilute, while its surrounding (outer medullary) interstitium becomes hypertonic. Since urea permeability of the thick ALH is also low, it cannot escape from the tubular lumen. As fluid moves into the late distal or connecting tubule, water is reabsorbed from the cortical and outer medullary collecting duct (CD) in the presence of ADH. These two segments of the CD, however, remain urea impermeable even in the presence of ADH. Therefore, the concentration of urea will have sharply increased by the time the tubular fluid reaches the inner medulla. At that point, ADH facilitates the reabsorption of both water and urea by the CD. Urea is "trapped" and accumulates in the medullary and papillary interstitium. Since the descending limb of the loop of Henle (DLH) is relatively impermeable to both NaCl and urea (Table I), urea will act osmotically to extract water from the descending limb, thus increasing the concentration of NaCl in luminal fluid above that in the interstitium. When tubular fluid now enters the water impermeable, NaCl permeable thin ALH, NaCl moves passively down its concentration gradient into the interstitium and renders the tubular fluid hypotonic to the surrounding interstitium. Since the thin ALH is also somewhat permeable to urea, urea will enter into this nephron segment and be available for its further concentration in the CD. Energy has therefore been transferred from the thick ALH (outer medulla) to the inner medulla and papilla in the form of a cencentrated urea solution.

The next question we have to pose is: How is the high solute concentration in the inner medulla/papilla region of the kidney preserved? This task is entrusted to the vasa recta. These blood vessels by means of precise tuning between the concentration process and blood flow fulfill a dual role—that of solute trapping by means of countercurrent exchange and that of removal of water which has been reabsorbed from the DLH and the CD.

TABLE I

PERMEABILITY CHARACTERISTICS OF SEGMENTS OF RABBIT RENAL TUBULES IN THE PRESENCE OF ADH

	Active Salt Transport	Permeability			
		H_2O	Urea	Na	Cl
Thin DLH	0	++++	±	±	±
Thin ALH	0	0	++	+++	++++
Thick ALH	++++	0	0	+	±
Convoluted DT	±	0	0	?	?
Cortical DC	++	++	0	?	?
Outer Med. CD	+	++	0	?	?
Papillary CD	+	++	++	?	?

Role of antidiuretic hormone

It is apparent from the above that ADH plays a pivotal role in this process and that urea reabsorption from the inner medullary CD, i.e. solute concentration in the inner medulla/papilla region of the kidney, will diminish as soon as circulating ADH concentrations decrease below a critical level. Osmotic water abstraction from the DLH will diminish, thereby causing an increased urine flow and further dilution of the urine. Under physiological conditions therefore, the shift from the concentrating state to the diluting state will be modulated mainly by ADH.

Mode of action of ADH

Exciting advances in renal physiological techniques, peptide chemistry and electron microscopy have profoundly advanced our understanding of the cellular action of ADH [arginine vasopressin (VP)]. While this short review is not the place to detail these newer findings (which are superbly described in a review by Dousa and Valtin[10]), a few comments are in order. VP has been shown to bind to a VP receptor in the baso-lateral membrane of the tubular cells. In this membrane a VP-dependent adenylate cyclase activates cyclic 3',5' AMP (cAMP), which in turn increases the water permeability of the luminal membrane of the tubular cell. This permeability increase is mediated by the phosphorylation of a membrane protein under the stimulation of cAMP dependent protein kinase. Water flow is facilitated in addition by a system of microtubules, which are unbranched, tubular structures, polymerized from a soluble protein called tubulin. These microtubules appear to

TABLE II

MECHANISMS REQUIRED FOR URINE CONCENTRATION

A. GENERATION AND MAINTENANCE OF OSMOTIC GRADIENT
 1. Sufficient NaCl delivery to medullary segments
 2. Sufficient NaCl transport in medullary segments
 3. Sufficient urea trapping in renal medulla
 a. Appropriate amounts of ADH circulating
 b. Appropriate response of collecting ducts to ADH
 4. Sufficient number of functioning nephrons
 5. Appropriate renal blood flow

B. UTILIZATION OF OSMOTIC GRADIENT
 1. Appropriate amounts of ADH circulating
 2. Appropriate response to ADH by collecting ducts
 3. Appropriate urine flow rate

TABLE III

POLYURIC SYNDROMES — CLASSIFICATION

A. ADH SUPPRESSED OR ABSENT
 1. ADH — secretion suppressed — primary polydipsia
 2. ADH — secretion impaired or absent — central diabetes insipidus

B. ADH SECRETION ADEQUATE
 1. Impaired generation and maintenance of osmotic gradient
 a. Hypokalemia
 b. Hypercalcemia
 c. Malnutrition
 d. Osmotic diuresis (glucose, urea, salt, mannitol, chronic renal failure)
 e. "Loop" diuretics
 f. Sickle cell disorders
 g. Hyperviscosity syndrome
 h. Tubulo-interstitial nephropathies (chronic pyelo, analgesic nephropathy, radiation nephritis, medullary cystic disease, hydronephrosis, non-obstructive urinary infections, Sjogren's Sx, transplant kidney, amyloid)
 2. Impaired utilization of osmotic gradient
 a. Impaired response of collecting duct to ADH
 Congenital nephrogenic diabetes insipidus
 Drugs (lithium carbonate, demeclocycline, colchicine)
 Hypercalcemia
 Anyloidosis
 b. Inappropriate flow rate of urine
 Osmotic diuresis
 Post-obstructive diuresis

increase water permeability of the luminal membrane and their disruption by vinea alkaloids (vinblastine) and colchicine will diminish it. Besides an increased water permeability of the luminal membrane, electron microscopical observations have demonstrated a marked widening of inter cellular spaces under the influence of VP. Attached to the lateral tubular cell membranes, limiting these spaces, are fibrous filaments, which are composed of proteins with biochemical properties similar to those of muscle actin. Contraction of these microfilaments possibly facilitates the dilation of the intercellular spaces, since their structural disruption by cytochalasins (antibiotics) dramatically impairs the effect of VP on the widening of intercellular spaces. As with a number of other hormonal effects, there are "long" and "short" feedback loops. A remarkable "short" one involves prostaglandin E_2 (PGE_2) as follows: VP stimulates the release of arachidonic acid from medullary cells which results in the increased formation of prostaglandin endoperoxides (PGEP) and PGE_2.[11] PGEP and possibly PGE_2 in turn inhibit VP dependent adenylate cyclase and therefore the VP induced formation of cAMP. Prostaglandin synthetase inhibitors (many non-steroidal anti-inflammatory drugs like ASA, indomethacin, ibuprofen, naproxen) will interrupt this feedback loop by diminishing the formation of both PGEP and PGE_2 and therefore enhancing the effect of VP.

Mechanisms required for urine concentration (Table II)

It is useful to analyze polyuric disorders by examining their impact on three mainstays in the formation of a concentrated urine, i.e., generation, maintenance and utilization of an osmotic gradient in the kidney. The loss or impairment of any one of the links in this chain will prevent the formation of a concentrated urine. As mentioned before, ADH plays a central role and it will serve as the cornerstone in the

Classification of Polyuric syndromes (Table III)

There are two important situations in which ADH is either suppressed by increased fluid intake, i.e., primary polydipsia or not available in sufficient amounts, the latter situation being referred to as *central diabetes insipidus* (CDI). According to Maffly,[12] the pathogenesis of this insufficiency might theoretically be divided into five categories: (a) abnormality of osmoreceptors, (a situation usually presenting with marked hypernatremia because receptors initiate the release of ADH too late.) (b) Abnormally low production of either VP or neurophysin, a hypothalmic transmitter hormone. (c) Defective release of VP. (d) Defective integration of the thirst-ADH system (i.e., thirst stimulation before ADH stimulation?) and (e) Accelerated degradation of VP or circulating antibodies against it. Analysis of clinical disorders, summarily referred to as CDI has not been profound enough to allow such a categorization. As our understanding of the above mechanisms increases we should be able to localize these clinically observed defects better. There are for example forms of CDI, which are accompanied by disturbances in the thirst sensation, closely situated to the para ventricular and supra optical areas in the hypothalamus, which control the release of ADH. Too little ADH with too little thirst is a dangerous combination which invariably leads to profound hypernatremia. The clinical causes for CDI are listed in Table IV. The most common is the acquired idiopathic type, (about 50% of all cases). The most dreaded complication of longstanding CDI besides severe dehydration is hydronephrosis. The treatment consists of replacement therapy, either as VP nasal spray or preferably as the longer acting Deamino-D-Arginine VP (DDAVP) or Deamino-valine-D-Arginine VP (DVDAVP) preparations.[13]

The second group of polyuric syndromes exists in the presence of adequate or increased amounts of circulating ADH and is caused either by faulty generation and/or maintenance of an osmotic gradient in the medulla or by a failure to utilize the osmotic gradient by the collecting duct. There are many situations, where this latter differentiation cannot be made, i.e.,

TABLE IV

CENTRAL DIABETES INSIPIDUS — CAUSES

A. PRIMARY
 1. Congenital (less than 1% of all cases)
 Autosomal dominant
 X-linked recessive
 2. Idiopathic (about 50% of all cases)

B. SECONDARY
 1. Neoplasms
 Craniopharyngioma
 Optic Glioma
 Pinealoma
 Melanoma
 Metastatic
 2. Trauma
 Basal skull fracture
 Neurosurgery
 3. Other
 Eosinophilic granuloma (Hand-Schüller-Christian Disease)
 Congenital malformations (Laurence-Moon-Biedl Syndrome)
 Infectious disorders (encephalitis)
 Vascular disorders (postpartum necrosis)

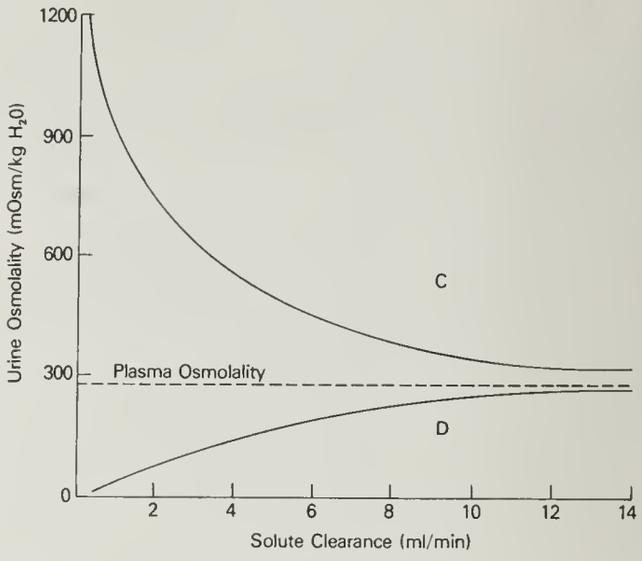


FIGURE 2
If solute excretion is high, urine concentration approaches isotonicity. In the concentration mode (C), urine concentrations diminishes, in the diluting mode (D), urine concentration increases as solute excretion is augmented.

a collecting duct which does not respond to ADH therefore not utilizing an osmotic gradient, is not efficient in the accummulation of urea in the inner medulla and therefore the generation of an osmotic gradient, either. The different causes listed in the table are therefore either listed twice or in the category where they appear to have the most effect.

Hypokalemia has been studied extensively in humans and experimental animals. It appears that mild hypokalemia does not affect the concentrating mechanism in the kidney, but leads to thirst stimulation, polydipsia, and secondarily to polyuria.[14] As potassium depletion progresses, there is impairment of NaCl reabsorption in the thick ALH (impairment of mitochondrial function in the outer medulla[15]). In addition, prostaglandin secretion is excessive and would be expected to impair the response of the CD to VP.[16] It is of interest that extracellular hypokalemia (at the level of 2.5mEq/L induced by metabolic alkalosis) does not lead to a concentrating defect.[17] Therefore it is probably the reduction of tissue potassium which is detrimental to the concentrating mechanism. The concentrating defect in *malnourished* patients is due to the lack of urea, which is therefore not available for recirculation in the medulla. Administration of urea in these patients corrects this abnormality.[18] *Non-reabsorbed substances* which increase the solute concentration of tubular fluid early in the nephron (proximal tubule, loop) interfere with water reabsorption, thereby increasing the concentration gradient of Na and Cl across the tubular cell, which in turn impairs the reabsorptive capacity of the loop, preventing the accumulation of solute in the medulla. These substances therefore not only increase urine flow, preventing utilization of an osmotic gradient, they also impair the generation of this gradient by interference with the "single effect" in the thick ALH. This situation is depicted in the upper curve of Figure 2. Most *diuretics* will interfere with the utilization of the osmotic gradient and therefore impair concentrating ability. The so-called "loop" diuretics (furosemide, ethacrinic acid) inhibit active chloride transport in the thick ALH, thereby interfering with the energy source for the countercurrent system and preventing the build up of an osmotic gradient in the kidney. They have been shown in animal experiments to dissipate the solute gradient between cortex and medulla within five minutes.[6] This demonstrates that generating and maintaining this gradient is a very dynamic process.

The concentrating defect in *sickle cell disease* relates principally to a disturbance in medullary bloodflow. This defect is reversible in children and adolescents (up to about age 15 years[19]) by the exchange transfusion of HbA blood. It is of interest that maximal urine concentrating ability is impaired much earlier and more severely than the reabsorption of solute free water (T^cH_2O), which shows a defect only late in the disease.[20] The following mechanism for the renal concentrating defect might therefore be proposed:

a. Sickling (due to hypertonicity and low medullary oxygen content) - hyperviscosity of blood - diminution of medullary blood flow - reabsorped H_2O not transported out of medulla - dilution of hypertonic interstitium (- diminished sickling).
b. Decrease in medullary blood flow - decreased O_2 delivery to medullary tubular cells - decreased NaCl transport.
c. Hypoxia (due to diminished blood flow) might also interfere with the generation of cAMP in vasopressin stimulated tissue.

Hypercalcemia affects both the generation and utilization of the osmotic gradient. Significant hypercalcemia is invariably accompanied by a decrease in GFR, causing diminished delivery of NaCl ("substrate") to the thick ALH, thereby diminishing the build up of a hypertonic interstitium.[21] In addition, there is calcification of medullary structures impeding the orderly performance of the countercurrent mechanism. There also is decreased cAMP formation under the stimulus of VP and a decrease of cAMP stimulated protein kinase.[22] Hypercalcemia also disrupts microtubules or prevents their polymerization from tubulin.[23]

The following disorders are primarily caused by an impaired utilization of the osmotic gradient in the kidney:

Congenital nephrogenic diabetes insipidus. This disease expresses itself during infancy and early childhood. Children present with a history of frequent need for diaper changes, dehydration and failure to grow. Diagnosis is suspected when the urine is examined—instead of being highly concentrated as would be appropriate, it is dilute or isotonic. While a defect in mice with hereditary nephrogenic diabetes in-

Educational Resource Center For Training In Occupational Safety And Health

The University of Arizona recently received a five-year grant from the National Institute of Occupational Safety and Health (NIOSH) for the establishment of an Educational Resource Center in Occupational Safety and Health.

The Center is one of nine regional projects funded by NIOSH and it will serve the Southwest. Nine colleges, the School of Health Related Professions, and the Toxicology Program of the University are contributors to the Center. The program will have two major focuses: 1) training of personnel in the core areas of occupational health; medicine, nursing, industrial hygiene, and safety and 2) outreach programs for other universities and community colleges in the southwestern states.

Dr. Herbert K. Abrams, Professor and Head of the Department of Family and Community Medicine, is the Director of the Center. There is an Executive Committee composed of representatives of the component programs of the Center and a Board of Advisors representing the professions, labor, management, educators, and others in the southwestern states. The Center is administered under the jurisdiction of the Vice President for Health Sciences. Programs will include training for degrees in the various fields, continuing education, and short refresher courses.

In recent years the problems of occupational health and safety have burgeoned. Many of the old well-known occupational diseases still remain and new ones are being discovered as industrial processes and chemicals proliferate. One study estimated that as many as 100,000 occupational disease deaths occur in the United States each year. Official figures record about 15,000 occupational deaths annually. Recent experiences have demonstrated carcinogenic and teratogenic effects of certain chemicals; impairment of fertility; effects on the central nervous system; behavioral symptoms; and many other protean manifestations of the impact of working conditions on health.

When potential hazards of work are combined with other elements in the environment such as cigarette smoking or emotional stress, the challenge to physicians and other health care providers is indeed an immense one.

Such developments, combined with the 1970 passage of the landmark Occupational Safety and Health Act (OSHA) have dramatized the shortage of skilled professional personnel available to deal with these problems. There is a great need for more occupational physicians, nurses, industrial hygienists, safety specialists, and toxicologists. There is also a need for better health education of workers and management personnel. It is hoped that the NIOSH-funded centers will contribute to filling the gap in personnel and thus help to reduce the toll of occupational diseases and accidents.

The Center brings together on-going offerings in occupational health and enables the University to augment these into a more systematic and comprehensive program. For example, the existing toxicology program will be assisted toward strengthening its work in occupational toxicology. The safety management course now funded by NIOSH at the College of Business and Public Administration is included in the new grant. The work on pesticides at the College of Agriculture, the program for noise measurement and control and prevention of hearing loss at the College of Fine Arts, and other existing efforts will be enhanced.

At the College of Medicine, the grant will make possible the addition of faculty in occupational medicine and epidemiology. The College of Nursing will be able to develop courses in occupational health nursing and the College of Engineering will employ its first faculty member in industrial hygiene engineering. The grant also will finance the salary of a resident in Occupational Medicine, a subspecialty of the American Board of Preventive Medicine.

Thus, the Educational Resource Center in Occupational Safety and Health promises to be a valuable asset to the southwestern region in furthering professional development and public awareness in occupational health and safety.

Herbert K. Abrams, M.D.
Department Head
Family and Community Medicine

Louis J. Kettel, M.D., Dean
College of Medicine

President
John F. Kahle, M.D.

President Elect
Richard L. Dexter, M.D.

Secretary
Derrill B. Manley, M.D.

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William B. McGrath, M.D.
Stanley H. Nordomo, M.D.
Murray Spotnitz, M.D.
John Stoner III, M.D.
William C. Weese, M.D.
Donald J. Ziehm, M.D.

What follows is from a "Pulmonary clinician turned editor" and is worthy of republishing. MBB.

Staying Is Harder Than Getting There

Regular exercise that improves general physical fitness may lessen the risk of a heart attack and may increase one's capability of dealing with illness should it occur. Therefore, although a desk-bound editor, I elected to engage in a program of physical conditioning, and from the many programs widely publicized, selected that recommended by Kenneth H. Cooper.[1] Jogging in the early morning was pleasant during the summer, and the anticipation of reaching a goal, modestly set at a 3-km run each morning in 17 minutes, made the effort seem easy and worthwhile. Friends and family remarked on my improved appearance, I felt fitter and more energetic, and having reached the preset goal, I felt some smug satisfaction. However, the lovely summer mornings gave way to the dark, cold, and wet mornings of late fall, and the realization finally dawned that in pursuing a goal, it is much harder to stay than to get there. If I were to keep what I had gained, there lay before me the prospects of doing calisthenics and running, day in and day out, in good weather and in bad, for the rest of my life. Physical fitness, like many other attainments, demands regular, continued effort.

This experience gave me a new insight into some of the problems of patient management, expecially into the care of those who are obese, smokers, or alcoholics. It is one thing to persuade an overweight patient to reduce; it is another thing to have them remain at that reduced level of weight. This is dramatically illustrated by the report of Johnson and Drenick.[2] In their study, 50% of the patients who had reduced their weight had regained it all during two to three years, and 90% had gained it all back after nine years. Similar problems are encountered in patients with emphysema who are advised to give up smoking and in alcoholics who are advised to give up drinking. Reaching the immediate goal of being a nonsmoker or a nondrinker is relatively easy compared with maintaining that status permanently.

Medical care often establishes goals that are transient because physician and patient are too easily satisfied or are unwilling to make a long-term commitment that will make the difference between episodic medical care and health maintenance.

My clinical experience leads me to be skeptical about the commitment most people have toward health if that commitment means regular exercise, a spartan diet, and abstinence from cigarettes and alcohol. It also makes me question the effectiveness of programs to detect and control hypertension if control requires a lifetime commitment to diet and daily medication. Should we strive in medicine to reach goals that cannot be maintained?

Physicians are well aware of the fact that staying is harder than getting there. To the medical student, the ultimate goal is to receive the MD degree and to be licensed. To the resident it is board qualifications followed by a successful practice. Having achieved these goals, the physician is then confronted with a lifetime devoted to journal reading, attendance at scientific meetings, and study for relicensure and recertification. The skills and knowledge that are sufficient for today will not suffice for tomorrow and will decline if not continually practiced and relearned. Medicine, like running, requires continuous practice and work if it is to be done well. Becoming a physician is relatively easy; it is staying there that is hard.

WILLIAM R. BARCLAY, MD

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1. Cooper, K.H.: **The New Aerobics**, New York, Bantom Books, Inc., 1970.
2. Johnson, D., Drenick, E.J.: Therapeutic fasting in morbid obesity. **Arch Intern Med** 137:1381-1382, 1977.

Review of the Hamer Education Loan Fund held priority at the mid-winter state board meeting and resulted in a proposal of discontinuance due to changing conditions since its formation in 1950.

Originally established as a student nurse loan fund to encourage recruitment into nursing, the statewide project has productively endured for longer than similar Auxiliary loan programs throughout the nation. Many capitulated after short duration, but our loan fund held steady through many changing demands and requirements in the nursing field.

Modified periodically to expand careers eligibility and adjust the financial regulations, the program has survived altered circumstances largely because of the efforts of dedicated committee members who have found it increasingly difficult to keep the revolving fund active.

After considering many factors, board members passed a recommendation to the general assembly scheduled to convene at the Annual Meeting in Tucson. If accepted, the proposal would discontinue the education loan fund with two conditions: 1) honor existing agreements, and 2) continue collection of outstanding loans. Granting of any new financial assistance would be by individual consideration and under policies not yet formed.

The diminishing number of requests plus collection difficulties have prompted the board recommendation. Change of career definitions, attitudes, costs, and business techniques over its 28-year span of existence has diluted the program's strength, making it vulnerable to re-evaluation and probable demise. We salute the gratifying success it achieved in meeting a professional need.

Mrs. M.W. (Jean) Phillips
Auxiliary editor

Correspondence

M. Block, M.D.
Editor

Dear Doctor Block:

Several months ago a colleague of yours, Dr. Gorrell, who had read my letter to the editor in May 30, 1977 issue of *JAMA* entitled "Dantrolene, Pelvic Neoplasm and Muscle Spasms: Report of A Case," wrote to me asking me that I update him on further experience I might have with dantrolene and that I submit a letter to you for possible publication in *Arizona Medicine* detailing my experiences with the drug. Because I had moved from Michigan to California in late June, his letter only reached me a couple of weeks ago. Though embarrased about the delay, I promptly replied to Dr. Gorrell.

At his suggestion I am enclosing a letter summarizing my experience with dantrolene, both with my original case, as well as with two subsequent cases. I hope you will find the information useful and worthy of inclusion in *Arizona Medicine*. I apologize to you also for the great delay, but as I indicated above it was due to circumstances beyond my control. Please contact me if I can be of any further assistance.

Yours truly,
Alan M. Myers, M.D.
Senior Fellow
Division of Hematology/Oncology

Dantrolene, Neoplasms, and Muscle Spasms: Experience with Three Patients

Malignant neoplasms can originate in, extend or metastasize to peripheral or central nervous tissue causing painful, debilitating skeletal muscle spasms. This probably occurs most commonly when the sacral plexus or pelvic peripheral nerves are involved by primary pelvic neoplasms, or by neoplasms metastatic to the pelvis, but also can occur when the spinal cord, brain, brachial plexus, and other peripheral nerves are similarly involved. Attempts to control these spasms and resulting pain with the older, centrally-acting skeletal muscle relaxants such as methocarbamol (Robaxin) and diazepam (Valium) have generally been unsuccessful. Narcotic anagesics often do not provide adequate control of spasm-induced pain. Summarized here are three cases of painful, skeletal spasms that we have successfully controlled.

Case Summaries:

The first patient had an adenocarcinoma of the rectum with extensive pelvic disease and pulmonary metastases.[1] Roentgenograms of the pelvis demonstrated sacral erosion by tumor; a pelvic echogram delineated a large pre-sacral mass. While on chemotherapy, he developed excrutiatingly painful muscle spasms in both lower extremities which abated only briefly and partially after irradiation of the pelvis. Diazepam, methocarbamol, and narcotic analgesics were ineffective at relieving the pain and spasms. Within one week of instituting dantrolene sodium therapy at the low dosage of 25 mg daily, the patient experienced complete relief of the spasms. Neither tolerance nor toxicity was evident after four months of treatment.

The next patient had an extensive ovarian carcinoma documented by surgery and ultrasound studies. She developed painful muscle spasms in her lower extremities which were not relieved by chemotherapy, radiation therapy, diazepam, methocarbamol or narcotic anagesics. Before a spinal nerve block was performed out of desperation, she was given a trial of dantrolene sodium at a dosage of 25 mg daily. Her symptoms disappeared within four days and had not recurred at the time of her death two months later. No drug tolerance or toxicity was detected.

The final patient had breast carcinoma with pelvic bone metastases demonstrated by roentgenograms. Over a period of several months she developed progressively more severe muscle spasms in her right lower extremity. A myelogram was non-diagnostic. During this period trials of several chemotherapy regimens were considered failures. Neurologic and neurosurgical consultants felt the cause of the spasms to metastatic tumor within the pelvis involving peripheral nerves. One week after therapy with dantrolene sodium was instituted at a daily dosage of 25 mg, the patient had obtained complete relief which has lasted 3½ months thus far. No toxicity or tolerance have been noted.

Comment

Dantrolene sodium, introduced in 1974, is a peripherally-acting skeletal muscle relaxant that acts directly on excitation-contraction coupling. It does not cause detectable anesthesia, analgesia, or impairment of reflexes or coordination, and there is no evidence that it interferes with neuromuscular transmission. Other excitable tissues do not seem to be affected. The majority of patients in whom the drug has been evaluated have had cerebral-vascular accident damage, spinal cord injuries, and cerebral palsy. Prolonged follow-up has shown no evidence of tolerance or toxic drug accumulation in these patients. The most common side effects have been drowsiness, dizziness, nausea and vomiting. The drug is metabolized in the liver, but only rare documented incidences of hepato-toxicity have been reported. An excellent review of the pharmacology of dantrolene was published by Dykes in 1975.[2]

In summary any patient who has painful or debilitating skeletal muscle spasms from a primary of metastatic neoplasm, regardless of location, warrants a therapeutic trial of dantrolene sodium. The drug may also have value in cases of intractable hiccoughs, regardless of etiology.

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"Out of Context" (Dry Gulch)
John W. Kennedy, M.D.

Cholesterol — the whipping boy for a decade or more may not be the "coronary artery plugger" as touted by "some scientific bodies and commercial interests." Ref. Practical Cardiology, November 1977.

Holistic: whence cometh and what meaneth this word when applied to medical problems? So far it hasn't made that best seller Webster's Unabridged Dictionary but it certainly is gaining status in medical literature. We hear of the holistic approach to the patient, of the holistic approach to the drug management of stable angina pectros. In some of the manners in which it is used, I am not sure whether it means a holy approach or a whole approach or whether indeed it is another black hole.

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

NOMINATING COMMITTEE

The meeting of the Nominating Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona on Saturday, February 4, 1978, a quorum being present, convened at 2:12 p.m., Edward Sattenspiel, M.D., Past President and Chairman, presiding.

NOMINATIONS

Following a review of the bylaws regarding nominating restrictions, the following slate of officers was compiled:

Vacancies in Office	Nominees
President-Elect (1978-79)	W.E. Crisp, M.D. L.J. Shapiro, M.D.
Vice President (1978-79)	J.T. Clymer, M.D. R.F. Dahlen, M.D.
Secretary (1978-79)	J.A. Ash, M.D. R.D. Zonis, M.D.
Treasurer (1978-79)	N.L. Robles, M.D. H.C. Tretbar, M.D.
Speaker of the House (1978-79)	R.A. Price, M.D. M.B. Block, M.D.
Editor-in-Chief (1978-79)	M.B. Block, M.D. R.A. Price, M.D.
Delegate to AMA (1/1/79-12/31/80)	A.V. Dudley, M.D. J.S. Whaley, M.D.
Alternate Delegate to AMA (1/1/79-12/31/80)	W.C. Scott, M.D. C.D. Christian, M.D.
Central District Director (1978-81) (Maricopa)	T.L. Mobley, M.D. P. Levy, M.D.
Central District Director (1978-81) (Maricopa)	H.N. Munhall, M.D. R.L. Collins, M.D.
Northeastern District Director (1978-81) (Apache, Gila, Navajo)	D.L. Neel, M.D. R.D. Martin, M.D.
Southern District Director (1978-81) (Pima)	J.M. Layton, M.D. G.D. Comerci, M.D.

Meeting adjourned 2:34 p.m.

FINANCE COMMITTEE

The meeting of the Finance Committee of the Arizona Medical Association, held at 810 W. Bethany Home Road, Phoenix, Arizona, on Saturday, February 18, 1978 convened at 10:01 a.m., John T. Clymer, M.D., Treasurer and Chairman, presiding.

1979 BUDGET

The committee reviewed the entire budget item by item making several changes in the process.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THE FOLLOWING BUDGET FOR 1979:

CONDENSED STATEMENT OF REVENUES AND EXPENDITURES

GENERAL DEPARTMENT	
Revenue	$ 595,605.00
Expenditure	578,400.00
	17,205.00
COMMITTEE DEPARTMENT	
Revenue	83,100.00
Expenditures	108,100.00
	(25,000.00)
ANNUAL MEETING	
Revenue	60,600.00
Expenditures	56,640.00
	3,960.00
PUBLISHING DEPARTMENT	
Revenue	72,470.00
Expenditures	68,430.00
	4,040.00
TOTAL ALL OPERATIONS	
Revenue	811,775.00
Expenditures	811,570.00
	$ 205.00

GENERAL DEPARTMENT

REVENUE		
401	AMA Commissions	$ 10,000.00
403	Dues-Active Members	465,980.00
404	Dues-Interns/Residents	250.00
406	Dues-Service Members	600.00
420	Printing Income	61,000.00
421	Sale of Supplies	500.00
429	Administrative Income	25,000.00
471-3	Building Assessment	8,875.00
474-5	Rental Income	23,400.00
	Total Revenue	$595,605.00
EXPENDITURES		
501	Auditing	$ 4,000.00
504	Dues, Subscrips, cont.	3,400.00
505	Equipment Maint. & Rent	18,000.00
507	General O/H (recovery)	(4,400.00)
508	Insurance, General	35,000.00
509	Insurance, Health	10,000.00
510	Legal	27,000.00
511	Meeting	28,000.00
512	Miscellaneous	3,000.00
513	Payroll Taxes	15,000.00
514	Postage	11,000.00
515	Printing	5,000.00
516	Refunds-ArMA Comm.	10,000.00
518	Salaries	247,000.00
519	Supplies	9,000.00
520	Telephone	12,000.00
521	Travel	15,000.00
522	President's Expense	10,000.00
530	Printing Dept. Expense	61,000.00
571	Depreciation Expense	19,000.00
572	Building Maintenance	8,000.00
574	Building Supplies	3,000.00
575	Interest	-0-
576	Property Taxes	7,000.00
577	Utilities	19,000.00
578	Building Insurance	3,000.00
	Total Expenditures	$578,400.00
		$ 17,205.00

COMMITTEE DEPARTMENT

REVENUE		
431	CME Processing Fee	$ 1,100.00
432	MICC Assessment	-0-
433	MICA Commission	80,000.00
481	Benevolent & Loan (Int.)	2,000.00
	TOTAL REVENUE	$ 83,100.00
EXPENDITURES		
500A	Articles of Inc. & Bylaws	$ -0-
500B	Benevolent & Loan	2,000.00
500D	Board of Directors	3,300.00
500E	Executive	1,700.00
500F	Finance	300.00
500H	Grievance	100.00
500I	History & Obituaries	-0-
500J	Maternal & Child Health	3,000.00
500K	Health Manpower	2,000.00
500L	Legislative	15,000.00
500M	Medical Education	5,000.00
500N	Medical Economics	1,200.00
500O	Occupational Health	-0-
500Q	Professional	3,000.00
500R	Public Relations	25,000.00
500S	Governmental Services	500.00
500U	House Staff	2,000.00
500W	ArMA Auxiliary	3,000.00
500X	Physicians Rehabilitation	1,000.00
500Y	Underwriting & Claim Review	40,000.00
500Z	MICC	-0-
	TOTAL EXPENDITURES	$108,100.00
		$(25,000.00)

ANNUAL MEETING DEPARTMENT

REVENUE		
441	Exhibit booth rental	$ 19,200.00
442	Sponsorship, speakers	2,600.00
443	Steak Fry	5,500.00
444	President's Banquet	4,000.00
445	Breakfast Panel #1	650.00
446	Breakfast Panel #2	650.00
447	Registration Fees	28,000.00
449		-0-
	Total Revenue	$ 60,600.00
EXPENDITURES		
541	Exhibit booth expense	$ 2,500.00
542	Guest speaker expense	10,000.00
543	Steak Fry	7,000.00
544	President's Banquet	7,500.00
545	Breakfast Panel #1	650.00
546	Breakfast Panel #2	650.00
547	Audio-visual equipment	2,500.00
548	Delegate's Manual	3,500.00
549	Employee hotel	2,500.00
550	Gratuities, hotel emp.	250.00
551	Miscellaneous	3,500.00
552	Newcomer Program	50.00
553	Nominating Committee	40.00
554	Postage	-0-
555	Printing	4,000.00
556	Promotion	4,000.00
558	Scientific Assembly Comte.	1,000.00
559	Supplies	2,000.00
560	Travel	400.00
561	Telephone	600.00
	Total Expenditures	$ 56,640.00
		$ 3,960.00

PUBLISHING COMMITTEE DEPARTMENT

REVENUE		
461	Advertising	$ 26,000.00
462	Reprints	2,000.00
463	Subscriptions, member	41,620.00
464	Subscriptions, nonmember	2,800.00
465	Other	50.00
	Total Revenue	$ 72,470.00
EXPENDITURES		
504	Dues and subscriptions	$ 130.00
505	Equipment maintenance	15.00
506	General Overhead	4,400.00
508	Insurance, general	225.00
509	Insurance, health	150.00
511	Meeting	100.00
512	Miscellaneous	-0-
513	Payroll Taxes	360.00
514	Postage	2,400.00
515	Printing	54,000.00
518	Salaries	6,000.00
519	Supplies	250.00
521	Travel	400.00
	Total Expenditures	$ 68,430.00
		$ 4,040.00

PRESIDENT/ PRESIDENT-ELECT EXPENSE

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THEY CONSIDER CHANGING THE PRESENT POLICY TO ONE THAT WOULD PROVIDE THE PRESIDENT A BUDGET OF UP TO $7,500 AND PROVIDE THE PRESIDENT-ELECT A BUDGET OF UP TO $2,500 PER YEAR.

COMMITTEE STRUCTURE REVIEW

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE EXECUTIVE COMMITTEE THAT THE FINANCE COMMITTEE IS CONCERNED ABOUT THE SIZE AND EFFECTIVENESS OF SOME OF THE ASSOCIATION'S COMMITTEES, AND THEY RECOMMEND THAT ALL COMMITTEE STRUCTURES BE REVIEWED.

PHYSICIAN PLACEMENT SERVICE

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE EXECUTIVE COMMITTEE THAT THEY CONSIDER PLACING A CHARGE OF UP TO $25 ON PROVIDING THE PHYSICIAN PLACEMENT SERVICE BULLETINS TO PHYSICIANS WHO ARE FROM OUTSIDE THE STATE OF ARIZONA.

LEGISLATIVE COMMITTEE

The meeting of the Legislative Committee of the Arizona Medical Association, Inc. held Saturday, January 14, 1978 at 810 W. Bethany Home Road, Phoenix, Arizona convened at 1:26 p.m., Selma E. Targovnik, M.D., Chairperson, presiding.

DEFINITION OF TERMS

Dr. Targovnik explained the following definition of terms which have been utilized the past few years in explanation of the Association's legislative activities:

ACTIVE SUPPORT: Means approval of bill and maximum staff time and financial expenditure (including legal counsel) will be made to achieve enactment.

GENERAL SUPPORT: Means approval of the principle of the bill, but that staff time and financial expenditures will be limited.

NO ACTION: Means no position has been taken one way or another on the bill.

NON-SUPPORT: Means disapproval of the bill, but that staff time and financial expenditure will be limited to defeat the bill.

ACTIVE NON-SUPPORT: Means disapproval of bill and that maximum staff time and financial expenditure (including legal counsel) will be expended to achieve defeat of the bill.

LEGISLATIVE REVIEW

MANDATORY BIRTH CERTIFICATE

The Board of Directors by letter of May 16, 1977 requesting the Legislative Committee to consider legislation which would require registration of births during a certain period of time. It was noted that the current statutes 36-322 provide for a certificate of

live birth for each child born alive in this state to be filed with the designated registrar within 7 days following such birth. IT WAS DETERMINED THAT ADDITIONAL LEGISLATION WAS UNNECESSARY.

REPEAL-LAY MIDWIVES

The Board of Directors during meeting held April 26, 1977 requested the Legislative Committee to work toward repeal of the present statutes providing for lay midwives. Considerable discussion ensued concerning current activities by the Department of Health Services in development of rules and regulations for lay midwives and that the Maternal and Child Health Committee of the Association was playing a major role in the development of such. It appeared to be the opinion of those working in public health as well as in the development of rules and regulations that this was a very inopportune time to seek repeal.

IT WAS MOVED AND CARRIED TO TAKE NO ACTION TOWARD REPEAL OF PRESENT STATUTES PROVIDING FOR LAY MIDWIVES.

MANDATORY INSURANCE MATERNITY BENEFITS

The Association introduced legislation during the last session providing for mandatory maternity benefits as in HB 2171.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT INTRODUCTION OF LEGISLATION PROVIDING FOR MANDATORY MATERNITY INSURANCE BENEFITS AS IN HB 2171 INTRODUCED DURING THE 1977 SESSION.

COMPULSORY DRIVERS EDUCATION COURSES [SB 1086 - 1977]

Dr. Raymond Grossman addressed the committee seeking support of the legislation to be introduced by Senator Sue Dye which will provide for compulsory driver education courses in the high school. Considerable discussion ensued.

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT LEGISLATION WHICH WOULD PROVIDE FOR COMPULSORY DRIVERS EDUCATION PROGRAMS WHICH WOULD INCLUDE ALL LICENSED MOTORIZED VEHICLES.

MERIT SYSTEM SELECTION FOR NEW APPOINTMENTS TO BOMEX

The Maricopa County Medical Society request that consideration be given to introducing legislation to provide for a merit system selection for new appointments to the Board of Medical Examiners. The Medical Practice Act was amended in 1976 to provide that the Governor "may" appoint BOMEX members from a list provided by the Medical Association as opposed to the previous "shall". The County Society suggested that the Medical Practice Act could be amended to mandate selection for new appointments to BOMEX by a merit system similar to the present method of nominating appellate court judges. The committee had available the constitutional provision of article 6-36 which provides for the appointments of appellate and trial court judges for consideration.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT THE CONCEPT OF "MERIT SYSTEM SELECTION FOR NEW APPOINTMENTS TO BOMEX" AND REFER THIS MATTER TO THE BOARD OF DIRECTORS FOR STUDY AND CONSIDERATION OF PROPER LEGISLATION TO ACCOMPLISH THE MERIT SYSTEM SELECTION.

PEER REVIEW

The Maricopa County Medical Society Board of Directors request that ArMA consider legislation to provide for additional protection for peer reviewers. It was stated that although MICA pays defendant physician cost, there is no deterrent to filing suit for disgruntled physicians who have been disciplined by their colleagues. If plaintiffs were required to pay defense cost of defendant committee members, frivolous suits filed only to assuage injured egos might be less likely.

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT THE DRAFTING, INTRODUCTION AND PASSAGE OF LEGISLATION PROVIDING FOR ADDITIONAL PROTECTION FOR PEER REVIEWERS BY REQUIRING PLAINTIFFS TO PAY DEFENSE COST OF DEFENDANT COMMITTEE MEMBERS IN PEER REVIEW ACTIVITIES WHEN SUIT IS CONSIDERED FRIVOLOUS.

GENERIC DRUG SUBSTITUTION

Legislation to provide for substitution of generic drugs for prescribed brand named drugs has been drafted for introduction. This legislation will provide that the pharmacist shall select a generic drug costing the patient less than prescribed drugs, that the name shall be included on the label, that it shall not be substituted if the prescriber indicated by any means that such a substitution is not to be made, and that it must be biologically, chemically and clinically equivalent to the brand name and has the same bio-availability of the brand name. It was noted that the Association has actively opposed such legislation in previous years.

IT WAS MOVED AND CARRIED TO TAKE NO ACTION ON LEGISLATION PROVIDING FOR SUBSTITUTION OF GENERIC DRUGS AS NOTED ABOVE.

CATASTROPHIC HEALTH INSURANCE PROTECTION

Draft legislation was reviewed providing for comprehensive and catastrophic health benefit protection. It was announced that this was an early draft of such legislation and consideration should be delayed until future drafts are available.

IT WAS MOVED AND CARRIED TO DEFER CONSIDERATION OF CATASTROPHIC HEALTH INSURANCE LEGISLATION EXCEPT TO ACTIVELY OPPOSE PROVISIONS IN THIS LEGISLATION PROVIDING FOR DIRECT BENEFITS TO PHYSICIAN'S ASSISTANTS AND WHERE ADVISORY COUNCILS ARE FORMED TO PROVIDE FOR PHYSICIAN REPESENTATIVES.

PROHIBIT AGE DISCRIMINATION

The AMA adopted a model state bill to prohibit age discrimination in employment practices and suggest that State Associations introduce such legislation in each state.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT THE INTRODUCTION OF MODEL LEGISLATION PROVIDED BY THE AMA TO PROHIBIT AGE DISCRIMINATION IN EMPLOYMENT PRACTICES.

PROHIBIT REBATES FOR REFERRALS OF PATIENTS

The AMA provided model State Legislation "to prohibit rebates for referrals of patients to health care providers or to certain facilities". It was noted that the Medical Practice Act provides that "giving or receiving, or aiding or abetting the giving or receiving of rebates, either directly or indirectly" is unprofessional conduct in the State of Arizona. Further that laboratory licensure laws provide for strict billing practices which include a prohibition of rebates.

IT WAS MOVED AND CARRIED TO TAKE NO ACTION CONCERNING AMA MODEL LEGISLATION PROHIBITING REBATES FOR REFERRALS AS CURRENT ARIZONA STATUTES ALREADY PROVIDE FOR SUCH PROHIBITIONS.

FISCAL NOTES

The AMA provides model state legislation which would require attachment of a fiscal note to all bills before any public hearing may be held or vote taken on such legislation. This fiscal note would contain a reliable estimate of the financial impact of legislation on state and local government and on the private sector.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT THE INTRODUCTION OF AMA MODEL LEGISLATION CONCERNING FISCAL NOTES.

METABOLIC SCREENING BILL

The Arizona Chapter of the American Academy of Pediatrics proposes that a metabolic screening bill be introduced into the next legislature in order to establish a program for screening genetic and metabolic disorders which are significant in terms of detectibility, cost effectiveness and long term significance. It was noted that the State of Maryland has a statute providing for such screening and is considered by the Pediatric community as model legislation. Concern was expressed as to the ability to properly prepare such legislation for the State of Arizona to consider on a timely basis. Legislation for this session must be introduced by February 6th.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT THE CONCEPT OF PROVIDING FOR METABOLIC SCREENING THROUGH LEGISLATION, THAT DURING THE INTERIM MEMBERS OF THE STATE DEPARTMENT OF HEALTH SERVICES, ARIZONA PEDIATRIC ACADEMY AND THE ASSOCIATION DRAFT SUCH LEGISLATION.

IMMUNIZATION REACTION LEGISLATION

The Arizona Chapter of the Academy of Pediatrics provided a copy of California legislation enacted in late 1976 that provides for reimbursement for injuries or severe reaction to immunizations which are required by law. This legislation does not apply to any accidents caused by negligence of the physician. The Pediatric Academy feels that this is model legislation which should be emulated in Arizona. It was noted that this was the first legislation of this nature enacted and that there was no experience to draw from. THE SUBJECT DIED FOR LACK OF MOTION.

HB 2084 - OPTOMETRY - USE OF

DIAGNOSTIC PHARMACEUTICAL AGENTS

Dr. Moore presented the new House Bill which would provide for use of diagnostic pharmaceutical agents by Optometrists as well as the House of Delegates resolution 4-77 adopted April 30th concerning this matter.

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT LEGISLATION PROVIDING FOR OPTOMETRISTS USE OF DIAGNOSTIC PHARMACEUTICAL AGENTS.

HB 2003 - LICENSING REGISTERED NURSE PRACTITIONERS

House Bill 2003 would provide for the licensing and practice of registered nurse practitioners providing exemptions to statutory provisions relating to the practice of medicine which would allow for the prescribing and dispensing of drugs as well as minor surgery.

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT HOUSE BILL 2003.

SB 1001 - SUNSET LAW

The Sunset Law introduced by Senator Osborn would provide for a joint legislative oversight committee to review certain regulatory agencies, one of which is the Arizona Board of Medical Examiners prescribing guidelines for hearings, reports and management analysis to determine its effectiveness and whether the agency should be terminated.

IT WAS MOVED AND CARRIED TO OFFER GENERAL NON-SUPPORT OF SUNSET LAW LEGISLATION.

SB 1002 - REPEAL OF VEHICULAR EMISSIONS INSPECTION LEGISLATION

This legislation would simply repeal the current legislation providing for vehicular emissions inspections. It was noted that the Board of Directors in meeting December 17th, reaffirmed its support of Arizona's vehicle emissions testing program originally stated in October 1976.

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT SENATE BILL 1002 REPEALING THE VEHICULAR EMISSIONS INSPECTION PROGRAM.

SB 1018 - POSSESSION OF DANGEROUS DRUGS

Current statutes provide that a person may lawfully possess prescription only drugs when held in the container in which it was delivered to him by the dispensing person. Legislation is proposed in which it "is a complete defense to prosecution for possessing such drug other than in the container in which it was delivered, that the amount of such drug is not more than that prescribed by a medical practitioner and that such a prescription was valid at the time of the alleged violation".

IT WAS MOVED AND CARRIED TO OFFER GENERAL SUPPORT TO LEGISLATION PROVIDING FOR A DEFENSE TO PROSECUTION FOR POSSESSING PRESCRIPTION ONLY OR DANGEROUS DRUGS IN OTHER THAN THE ORIGINAL CONTAINER.

HB 2002 - REPEAL - MEDICAID

Legislation has once again been introduced to officially repeal Medicaid from the Arizona statutes. The most current position of the Arizona Medical Association concerning "Medicaid" is as adopted by the House of Delegates on May 1, 1976 as follows:

RESOLVED, That the Arizona Medical Association actively encourage our legislative bodies and state health department organizations to scrutinize all pending Medicaid legislation in a manner which recognizes geographic needs and the adequacies or inadequacies of existing local systems of medical care.

IT WAS MOVED AND CARRIED TO TAKE NO ACTION ON HOUSE BILL 2002 PROVIDING FOR THE REPEAL OF MEDICAID.

HB 2023 - COUNTY PARTICIPATION IN EMERGENCY PARAMEDIC PROGRAMS

State law currently provides that a person other than an indigent shall not receive public aid by the county. House Bill 2023 prescribes a clarification of the authority of the county to participate in emergency paramedic programs when immediate hospitalization is necessary for the preservation of life or limb.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT LEGISLATION PRESCRIBING CLARIFICATION OF THE COUNTY'S AUTHORITY TO PARTICIPATE IN EMERGENCY PARAMEDIC PROGRAMS.

SB 1029 - MEDICAL CERTIFICATE OF COSMETOLOGIST, MANICURIST AND OPERATORS OF SUCH SHOPS

Current law provides that cosmetologist and manicurist must submit to their Board of licensure medical certificates. It is proposed that the Board of Cosmetology adopt and enforce reasonable rules and regulations requiring those who display symptoms of pulmonary tuberculosis to submit such test or examinations as a licensed physician deems appropriate.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT SENATE BILL 1029.

HB 2096 - MENTAL HEALTH LAW REVISION

Dr. Campbell introduced HB 2096 to the committee informing that this is principally the same legislation introduced last year to make certain corrections to the mental commitment legislation enacted in 1976, plus revising certain definitions which would clarify the commitment and treatment provisions.

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT THE MENTAL HEALTH LAW REVISIONS AS PROVIDED IN HOUSE BILL 2096.

HB 2086 - EXEMPTING PURCHASE OF INSULIN AND SYRINGES FROM SALES TAX

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT LEGISLATION PROVIDING FOR THE EXEMPTION OF SALES TAX FOR THE PURCHASE OF INSULIN AND SYRINGES.

HB 2076 - INTERNATIONAL WHEELCHAIR SYMBOL

This legislation would require the certain disability test be satisfied for persons to be issued number plates bearing the international wheelchair symbol, in addition to certificate issued by a physician.

IT WAS MOVED AND CARRIED TO GENERALLY NON-SUPPORT HOUSE BILL 2076 CONCERNING THE INTERNATIONAL WHEELCHAIR SYMBOL.

HB 2073 - APPROPRIATIONS FOR THE JOINT BOARD OF MEDICAL AND OSTEOPATHIC EXAMINERS

Mr. Cerf explained to the committee that last years legislation providing for a Joint Board of Medical and Osteopathic Examiners did not include an appropriation. Although the Joint Board has met on several occasions and rules and regulations enacted to provide for Physician's Assistants, continued operation is unlikely unless there is an appropriation to permit it.

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT THE JOINT BOARD OF MEDICAL EXAMINERS AND OSTEOPATHIC EXAMINERS SEEKING PROPER APPROPRIATIONS FOR ITS CONTINUATION.

ADVERTISING

Dr. Dexter reported that the Ad Hoc Committee on U.S. Supreme Court Decision Regarding Advertising met earlier on January 14th and recommend to the Legislative Committee that the Association propose legislation during this session of the legislature to amend the medical practice act by providing for a new definition of advertising under unprofessional conduct to read "Advertising a false, deceptive or misleading manner, advertising the quality of medical services, advertising by in-person solicitation, and advertising procedures and practices which are illegal".

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT LEGISLATION AMENDING THE MEDICAL PRACTICE ACT PROVIDING FOR A NEW DEFINITION OF ADVERTISING UNDER UNPROFESSIONAL CONDUCT TO READ "ADVERTISING A FALSE, DECEPTIVE OR MISLEADING MANNER, ADVERTISING THE QUALITY OF MEDICAL SERVICES, ADVERTISING BY IN-PERSON SOLICITATION, AND ADVERTISING PROCEDURES AND PRACTICES WHICH ARE ILLEGAL".

LAETRILE

It was noted that additional legislation on Laetrile had been introduced which would replace 1977 legislation by adding language specifically authorizing anyone to make or use Laetrile in Arizona, but forbidding manufacture in Arizona for sale out of state. House Bills would be forbidden to restrict the use of Laetrile and doctors and pharmacists could not be penalized for prescribing it.

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT LEGISLATION INVOLVING THE USE OF LAETRILE.

87th Annual Meeting in '78

Plan to attend the 1978 Annual Meeting April 25-29, 1978 at the Tucson Community Center and Marriott Hotel —

Mark your calendar.

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education.

Arizona Heart Institute
Arizona Medical Association, Inc.
Arizona State Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates
Phoenix Indian Medical Center
Phoenix Psychiatric Council
Maricopa County General Hospital, Phoenix
Memorial Hospital, Phoenix
St. Luke's Hospital & Medical Center, Phoenix
St. Joseph's Hospital & Medical Center, Phoenix
Tucson Hospitals Medical Education Program, Tucson
U of A Health Sciences Center
Veterans Administration Center, Prescott
Veterans Administration Hospital, Phoenix

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

Individual program listings below indicate Category of CME. Where not indicated, no credit has been approved. For further information on any programs, contact person named.

APRIL

PEDIATRIC RADIOLOGY FOR THE GENERAL RADIOLOGIST
April 14-16, 1978, Doubletree Inn at Scottsdale Mall, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contact: A.R. Sandrock, M.D., Chairman, Dept. of Radiology, Maricopa County General Hospital, 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 15 required hours toward the ArMA Certificate in Continuing Medical Education.

NEW HORIZONS FOR THE HANDICAPPED CHILD
April 22, 1978, Arizona State University, Tempe, AZ. Sponsor: Arizona Children's Hospital. Contact: Ms. Wendy Howard & Julia M. Luce, M.D., Student Health Service, Arizona State University. Approved for 7 category 1 hours.

ARIZONA MEDICAL ASSOCIATION 86th ANNUAL CONVENTION.
April 25-29, 1978, Tucson Community Center and Marriott Hotel, Tucson, AZ. Sponsor: Arizona Medical Association, Inc. Contact: Mr. Bruce E. Robinson, 810 W. Bethany Home Rd. Phoenix, AZ 85013. Approved for required hours toward the ArMA Certificate in Continuing Medical Education. Detailed program will appear in the March issue of *Arizona Medicine*.

AMBULATORY PEDIATRIC ASSOCIATION, INC. 18TH ANNUAL MEETING
April 24, 25, 1978. New York Hilton, N.Y., N.Y. Sponsor: Ambulatory Pediatric Association, Inc. Contact: Roberta Weiss, 4525 E. San Francisco St., Tucson, AZ 85210. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

ARIZONA UROLOGICAL SOCIETY LUNCHEON
April 27, 1978, 12 Noon, Marriott Hotel, Tucson, AZ. Sponsor: Arizona Urological Society. Contact: Everett D. Hendricks,

M.D., 1055 Ruth Street, Prescott, AZ 86301. Murray Feldstein, M.D., 900 N. San Francisco St., Flagstaff, AZ 86001.

INTERVENTIVE RADIOLOGY OF THE UPPER URINARY TRACT
April 29, 1978. Westward Look Hotel, Tucson, AZ. Sponsor: Arizona Radiological Society. Contact: Tim B. Hunter, M.D., U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 hour of Category 1 credit.

MAY

UPDATE: PRIMARY CARE 1978
May 3-6, 1978. Arizona Health Sciences Center Sponsor: U of A Continuing Medical Education. Contact: A. Vuturo, M.D., Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 25 required hours toward the ArMA Certificate in Continuing Medical Education.

COMBINED GROUND ROUNDS
May 3, 1978, Scottsdale Memorial Hospital. Sponsor: Family Practice Residency Programs of St. Joseph's, Good Samaritan, and Scottsdale Memorial Hospitals. Contact: Ann Larsen, R.N., Program Coordinator, Family Practice Residency Program, St. Joseph's Hospital and Medical Center, Phoenix, AZ 85013. Approved for required hours toward the AMA Certificate in Continuing Medical Education.

RURAL HEALTH/EMERGENCY CARE CONFERENCE
May 12, 13, 1978. Little America, Flagstaff, AZ. Sponsor: U of A Continuing Medical Education Program. Contact: John Hughes, M.D., Emerg. Serv. Dept., or Anthony Vuturo, M.D., Continuing Medical Education and Outreach, U of A Health Sciences Center. Tucson, AZ 85724. Approved for 12 required hours toward the ArMA Certificate in Continuing Medical Education.

SYMPOSIUM ON RADIATION PATHOLOGY
May 22, 23, 24, 1978. Wesward Look Resort, Tucson, AZ. Sponsor: U of A Health Sciences Center. Contact: David White, M.D., Anthony F. Vuturo, M.D., UAHSC, 1501 N. Campbell Ave., Tucson, AZ. Approved for 18 required hours toward the ArMA Certificate in Continuing Medical Education.

JUNE

17th ANNUAL STATE CONVENTION
Maricopa Chapter, American Association of Medical Assistants, June 9, 10, 11, 1978, Doubletree Inn, Scottsdale, AZ. Sponsor: Maricopa Chapter, American Association of Medical Assistants. Contact: Jan Neal, CMA Convention Chairman, 4502 N. 36th St., $108, Phoenix, AZ 85018.

MEDICAL HORIZONS: ADVANCES IN THERAPEUTICS
June 30-July 2, 1978. Little America Hotel, Flagstaff, AZ. Sponsor: Office of Continuing Medical Education, U of A Health Sciences Center, Tucson, AZ. Contact: Anthony F. Vuturo, M.D., AHSC, Tucson, AZ 85724. Approved for 10 required hours toward the ArMA Certificate in Continuing Medical Education.

JULY

21st ANNUAL RUIDOSO FAMILY PRACTICE SEMINAR
July 17-20, 1978. Headquarters Inn, of the Mountain Gods at Mescalero, New Mexico. Sponsors: New Mexico Chapter of the American Academy of Family Physicians. Contact: Bob Reid, Convention Dir., P. O. Box 456, Sunland Park, N.M. 88063. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

FIRST ANNYAL SUMMER PEDIATRIC CONFERENCE PREPARATION FOR RECERTIFICATION
July 7, 8, 9, 1978. 7:30 a.m.-2 p.m., Little America, Flagstaff, AZ. Sponsor: CME, U of A Health Sciences Center. Contact: Anthony F. Vuturo, M.D., ASHSC, 1501 N. Campbell Ave., Tucson, AZ 85724. Approved for 17.5 required hours toward the ArMA Certificate in Continuing Medical Education.

MONTHLY OR WEEKLY

HEADACHE ROUNDS
Tuesday afternoons or Thursday mornings weekly. Sponsor: Headache Group of the Southwest. Contact: Peggy Voigt, 2610 W. Bethany Home Rd., Phoenix, AZ 85017, or phone 242-6646.

SHRINE MEDICS MEETING
Second Tuesday of each month, Doctors Hospital, 5:45 p.m., J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

OFFICE PSYCHIATRY FOR THE PRIMARY PROVIDER
2nd Monday of month, 4811 N 7th Street Phoenix, AZ Sponsor Arizona Health Plan Contact T R Bittker M D Box 5000 Phoenix AZ 85010 Approved for required hours toward the ArMA Certificate in Continuing Medical

COUNTER TRANSFERENCE GROUP
Weekly Thurs 8-10 p m Sponsor Phoenix Psychiatric Council Contact James E Campbell, M D 5051 N 34th St Phoenix, AZ 85018 Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education

MONTLHY MEETING OF TUCSON RADIOLOGISTS
Last Tues of Month Plaza International Tucson, AZ Sponsor U of A Medical Center, Dept of Radiology Contact Irwin M Freundlich, M D., Arizona Medical Center, Dept of Radiology, Tucson, AZ 85724 Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education

PEDIATRIC GRAND ROUNDS
Tuesday 7:30 - 8:30 a.m. in Phoenix: 1st Tue. - Phoenix Indian Hospital, 2nd Tue. - Maricopa County Hospital, 3rd Tue. - Good Samaritan Hospital, 4th Tue. - St. Joseph's Hospital
Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

REVIEW OF FORENSIC PATHOLOGY, CURRENT CASE, SPECIAL TOPICS
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists, Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave. Phoenix, AZ. Approved for 1 required hour per session toward ArMA CME Certificate.

Arizona State Hospital

STAFF EDUCATION CONFERENCE
Wednesdays Weekly 1 p m Arizona State Hospital, Phoenix AZ Sponsor Arizona State Hospital Contact Howard E Wulsin M D Arizona State Hospital 2500 E Van Buren Phoenix AZ 85008 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

Baptist Hospital

SURGICAL GRAND ROUNDS
4TH TUESDAY OF EACH MONTH
Hospital Auditorium Baptist Hospital Phoenix Sponsor Baptist Hospital Phoenix Contact James B Shields M D 6036 N 19th Ave Phoenix AZ 85015 Approved for 1-1/2 required hours per month toward the ArMA Certificate in Continuing Medical Education

Barrow Neurological Institute

MEDICAL EDUCATION
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center. 350 W. Thomas Road, Phoenix, AZ 85013. Sponsor: St. Joseph's Hospital & Medical Center. Contact: John R. Green, M.D.,

Approved for one hour required credit toward the ArMA Certificate in Continuing Medical Education.

NEUROLOGY TEACHING CONFERENCE
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

NEUROSURGERY MORBIDITY CONFERENCE
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.

SPINAL INJURY CONFERENCE
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Room—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists.

NEUROPATHOLOGY OF GROSS SPECIMENS CONFERENCE
Thursdays, 7:30-8:30 a.m. in the Morgue.

NEUROLOGY-NEUROSURGICAL CLINICAL-NEURORADIOLOGICAL AND NEUROPATHOLOGICAL CONFERENCE
Fridays, 8-9 a.m., First Floor Conference Room.

NEUROPATHOLOGY OR NEURORADIOLOGY CONFERENCES
Fridays, 9 a.m., Neuropathology in Neuropathology Conference Room, Neuroradiology in First Floor Conference Room.

NEUROSURGICAL JOURNAL CLUB
Saturdays, 9-11 a.m., in Eighth Floor Conference Room.

Camelback Hospital

PATIENT STAFFING CONFERENCE
Three times weekly Camelback Hospital Phoenix, AZ Sponsor Camelback Hospital Contact Medical Director, Camelback Hospital, 5055 N 34th St, Phoenix, AZ 85018 Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

Good Samaritan Hospitals

DESERT SAMARITAN HOSPITAL
Wednesday Evenings 7 p m Sponsor Desert Samaritan Hospital Contact L A Rosati, M D, Laboratory, Desert Samaritan Hospital, Mesa AZ 85202 Approved for required hours toward the ArMA Certificate in Continuing Medical Education

CLINICAL CANCER CONFERENCE
3rd Wednesday every month, Butler Bldg Conference Room Good Samaritan Hospital, Phoenix, AZ Sponsor Good Samaritan Hospital Contact John A Bruner M D, 926 E McDowell Road, Phoenix, AZ 85006 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

PULMONARY DISEASE GRAND ROUNDS
Mondays – 12 Noon D-5 North Conference Rm Good Samaritan Hospital, Phoenix, AZ Sponsor Pulmonary Disease Teaching Service, Good Samaritan Hospital Contact Bernard E Levine, M D, Pulmonary Function Laboratory Good Samaritan Hospital, 1033 E McDowell Hospital, Phoenix, AZ 85006 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

CONTINUING MEDICAL EDUCATION PROGRAM
Maryvale Samaritan Hospital, every other Wednesday, 7-8 a.m., January 25, 1978 through December 27, 1978. Sponsor: Medical Staff of Maryvale Hospital. Contact: Richard C. Christensen, M.D. Approved for one required hour per session toward the ArMA Certificate in Continuing Medical Education.

Memorial Hospital, Phoenix

MONTHLY MEDICAL EDUCATION SEMINAR
Third Monday of the Month, 6 30 p m, Kiva Conference Room, Phoenix Memorial Hospital Sponsor Medical Staff of Memorial Hospital Contact George Scharf, M D, 1201 South 7th Avenue, Phoenix, AZ 85007 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

TUMOR BOARD CONFERENCE
Every Friday at Noon, Kiva Conference Room, Phoenix Memorial Hospital Sponsor Phoenix Memorial Hospital Contact Howard Kimball, M D, 333 West Thomas Road, Phoenix, AZ 85013 Approved for credit toward the ArMA Certificate in Continuing Medical Education

Scottsdale Memorial Hospital

CLINICAL PATHOLOGICAL CONFERENCE
4th Monday, Monthly, 12 45 p m, Scottsdale Memorial Hospital Scottsdale, AZ Sponsor Medical Staff Contact R C Good M D, Director of Medical Education, 7300 E. 4th St, Scottsdale AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

FAMILY PRACTICE CONFERENCE
1st Monday, Monthly, 12 45 p m, Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor Medical Staff Contact R C Good, M D, Dir of Medical Education, 7300 E 4th St, Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

MEDICAL GRAND ROUNDS
3rd Monday, Monthly, 12 45 p m, Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor Medical Staff Contact R C Good, M D, Dir of Medical Education, 7300 E 4th St, Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

MORBIDITY & MORALITY CONFERENCE
2nd Monday, Monthly, 12 45 p m, Scottsdale Memorial Hospital, Scottsdale, AZ Sponsor Medical Staff Contact R C Good, M D, Dir Medical Education, 7300 E 4th St, Scottsdale, AZ Approved for 1 elective hour per conference toward the ArMA Certificate in Continuing Medical Education

St. Mary's Hospital, Tucson

CARDIOLOGY CONFERENCE
Weekly – Friday 8-9 a m, St Mary's Hospital Auditorium, Tucson, AZ Sponsor St Mary's Hospital Contact A L Forte, M D, St Mary's Hospital, Tucson, AZ 85724 Approved for one required hour toward the ArMA Certificate in Continuing Medical Education

GRAND ROUNDS
Each Thursday 7 a m -8 a m, St Mary's Hospital and Health Center, Sponsor Depts. of Medicine, Surgery, Radiology, Pathology and Family Practice Contact Richard Silver, M D, Chairman, Medical Education and Library Committee, Century Medical Plaza, Suite 160, 1701 West St Mary's Road Tucson, AZ 85703 Approved for 1 required hour per round toward the ArMA Certificate in Continuing Medical Education

MONTHLY SPECIALTY CONFERENCE OF THE DEPARTMENT OF SURGERY
First Monday, each month, 7:30 a.m., Century Room A. Sponsor: St. Mary's Hospital & Health Center. Contact: Medical Staff Office, St. Mary's Hospital & Health Center, P.O. Box 5386, Tucson, AZ 85703. Approved for one required hour per session toward the ArMA Certificate in Continuing Medical Education.

U of A Health Sciences Center

CLINICAL IMMUNOLOGY, ALLERGY AND RHEUMATOLOGY ROUNDS
Every Friday Noon-1 p m Sponsor U of A College of Medicine, Dept of Internal Medicine, Clinical Immunology Section Contact John Boyer M D, U of A College of Medicine, Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

CEREBROVASCULAR DISEASE CONFERENCE
Mondays 5-6 p.m., weekly, Rm 5505 Surgery, U of AHSC. Sponsor: Depts. of Surgery, Neurology and Radiology, UAHSC. Contact: Dr. Wesley Moore, U of A Health Sciences Center, Tucson, AZ 85724.

ENDOCRINOLOGY SEMINAR
Every Thursday, Noon-1 p m, 1st, 3rd & 5th Thursday – Rm N318, VA Hospital, 2nd & 4th

Thursday Rm 6505 Tucson Medical Center Sponsor U of A College of Medicine Department of Internal Medicine Department of Internal Medicine Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

HEMATOLOGY-ONOCOLOGY CLINICAL CONFERENCE
Every Tuesday Noon-1 p m 1st 3rd & 5th Tuesdays – Rm 6505 AZ Medical Center 2nd & 4th Tuesdays – Rm N318 Veterans Adm Hospital Sponsor U of A College of Medicine Dept of Internal Medicine Contact Sidney Salmon, M D , U of A College of Medicine, Tucson AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

MEDICAL GRAND ROUNDS
Every Wednesday Noon-1 p m 1st 3rd & 5th Wednesday – Staff Conf Rm , VA Hospital 2nd & 4th Wednesday – Rm 5403 Arizona Medical Center Sponsor U of A College of Medicine, Dept of Internal Medicine Contact Jay Smith, M D , U of A College of Medicine Tucson, AZ 85721 Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education

NUCLEAR MEDICINE
Thursday, 7:30 a.m., Radiology Conf. Rm., AHSC, Tucson. Sponsor: Dept. of Diagnostic Radiology. Contact: Irwin M. Freundlich, U of A Health Sciences Center, Tucson, AZ 84724. Approved for one required hour per session toward ArMA CME Certificate.

NEUROLOGY GRAND ROUNDS
Every Friday, 12 noon. 1st, 3rd & 5th Fridays, U of A Health Sciences Center, Rm. 7505. 2nd and 4th Fridays, Veterans Administration Hospital. Sponsor: U of A Health Sciences Center, Dept. of Neurology. Contact: William A. Sibley, M.D., Dept. of Neurology U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

NEUROPATHOLOGY REVIEW CONFERENCE
Thursdays, 8:30 a.m., Rm 5120 Arizona Health Sciences Center. Sponsor: U of A College of Medicine. Contact: Peter C. Johnson, M.D., 1501 N. Campbell Ave., Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

NEUROSCIENCES SEMINAR
Every Friday, 7:30 a.m. U of A Health Sciences Center, Rm. 7505, Tucson, AZ. Sponsor: Dept. of Neurology. Contact: Colin R. Bamford, M.D. & Jose F. Laguna, M.D., Dept. of Neurology, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PATHOLOGY SEMINAR
Friday, 4:30-5:30 p.m., Rm 5120 AHSC. Tucson, AZ. Sponsor: Dept. of Pathology, Contact: Dr. Kenneth Ryan, Dept. of Path. U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour per session toward the ArMA Continuing Medical Education Certificate.

TUCSON PATHOLOGIST CONFERENCE
First Monday of the month, 7:30-9 p.m., Rotation Between Tucson Pathology Conf. Rms., Sponsor: Tucson Pathologists. Contact: Dr. Merle Clard, U of A Health Sciences Center, Dept. of Path., Tucson, AZ 84724. Approved for 1 required hour per session toward the ArMA Certificate in CME.

PEDIATRIC GRAND ROUNDS
Every Tuesday, 12:30 p.m. - 1:30 p.m. Alternates between Tucson Medical Clinic and U of A Health Sciences Center. Sponsor U of A College of Medicine, Dept. of Pediatrics. Contact: Hugh Thompson, M.D., 1501 N. Campbell, Tucson, AZ. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

PROBLEM CASE WORKSHOPS
3rd Monday of each month 7 30 a m Room 4410, Arizona Medical Center, Tucson, AZ Sponsor Division of Ophthalmology, U of A College of Medicine Contact H E Cross M D , Ph D , Arizona Medical Center, Dept of Surgery, Tucson, AZ Approved for 2 required hours toward the ArMA Certificate in Continuing Medical Education

PSYCHIATRIC GRAND ROUNDS
Every Wed Sept to May 4-5 30 p m Rm 8403, Arizona Medical Center Tucson, AZ Sponsor U of A College of Medicine Dept of Psychiatry Contact Alan Levenson, M D , U of A College of Medicine, Tucson, AZ 85721 Approved for 1-1/2 required hour per session toward the ArMA Certificate in Continuing Medical Education

CHEST RADIOLOGY
Monday, 5-6 p.m., weekly, Rm. 1535F, AHSC, Tucson, AZ. Sponsor: Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of Radiology, Tucson, AZ 85724. Approved for 1 required hour per session toward ArME CME Certificate.

GASTROINTESTINAL RADIOLOGY
Mondays, 7:30 a.m., weekly except for summer months. Sponsor: Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., Diagnostic Radiology, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 equired hour toward ArMA CME Certificate.

RADIATION ONCOLOGY PLANNING CONFERENCE
Friday, weekly, 8:30-10 a.m., AHSC Rm. 0655. Sponsor: Radiation oncology Section, UAHSC, Tucson, AZ. Contact: Robert S. Heusinkveld, M.D., Ph.D., U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1.5 required hours per session toward ArMA Certificate in CME.

RADIOLOGY-RHEUMATOLOGY CONFERENCE
Thursday, 8 a.m., UAHSC, Library, Rm. 1535C. Sponsor: Dept. of Radiology, Contact: Irwin M. Freundlich, U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour toward the ArMA Certificate in CME.

VASCULAR RADIOLOGY
January thru June 1978. 7:30 a.m. to 8:30 a.m. every Friday. Dept. of Radiology. Sponsor: Dept. of Radiology, U of A Health Sciences Center. Contact: I. M. Freundlich, M.D., U of A Health Sciences Center Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

RADIOLOGY INTERESTING CASE CONFERENCE
Thursday, noon, Veterans Administration Hospital, Tucson, AZ. Sponsor: U of A Health Sciences Center, Tucson, Radiology Dept. Contact: Irwin M. Freundlich, M.D., U of A health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA CME Certificate.

RESIDENT'S CONFERENCE
Wednesday/weekly 5-6 p.m., Diag. Radiology Conf. Rm., U of A Health Sciences Center. Sponsor: U of A Health Sciences Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour per session toward the ArMA Certificate in Continuing Medical Education.

TRAUMA CONFERENCE
Every Thursday, 4-5:00 p.m. Health Sciences Center, Rm. 5505, Tucson, AZ. Sponsor: U of A College of Medicine, Dept. of Surgery. Contact: Martin Silverstein, M.D., Dept. of Surgery, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1 required hour per session toward the ArMA Certificate in Continuing Medical Education.

GRAND WARD ROUNDS – TRAUMA
Every Tuesday, 8 a m Arizona Medical Center, Tucson AZ Sponsor U of A College of Medicine Surgery Dept , Trauma Section Contact Martin Silverstein, M D U of A College of

Director. Approved for one hour required credit toward the ArMA Certificate In Continuing Medical Education.

MONTHLY MEETING TUCSON ULTRASONOGRAPHY GROUP
First Wednesday of month, 4:30-5:30 p.m., Arizona Health Sciences Center. Sponsor: Dept. of Diagnostic Radiology. Contact: Irwin M. Freundlich, M.D., AHSC, Tucson, AZ 85724. Approved for 1 required hour per session toward ArMA CME Certificate.

TUCSON UROLOGICAL SOCIETY SCIENTIFIC MEETING
Monthly, Sponsor: Depts of Surgery & Urology, AHSC, Tucson. Contact: George W. Drach, M.D., U of A Health Sciences Center, Tucson, AZ 85724. Approved for 1.5 required hours per session toward ArMA CME Certificate.

GENERAL UROLOGY CONFERENCE
Tuesday 5-6 p.m., Thursday noon-1:30 p.m. AHSC Meeting rms., & VA Hospital. Sponsor: Dept of Surgery/Urology Section. Contact: George W. Drach, M.D. Dept. of Surgery/Urology Section, U of A Health Sciences Center, Tucson, AZ 85724. Approved for one required hour toward ArMA CME Certificate.

VA Hospital, Phoenix

CANCER SYMPOSIUM (fomerly Tumor Board)
Each Wednesday, 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

CARDIOLOGY CONFERENCE
2nd Thursday 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail M.D., VA Hospital 7th & Indian School Rd. Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL-SURGICAL CHEST CONFERENCE
1st and 3rd Thursday, 1 p.m., T-5 Sponsor VA Hospital Phoenix. Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

CLINICOPATHOLOGY CONFERENCE
4th Thursday of 3rd Mo. (Mar., June, Sept & Dec.), 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Education.

DERMATOLOGY CONFERENCE
1st, 2nd & 3rd Wednesday Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

HEPATOLOGY CONFERENCE
2nd and 4th Tuesday, 1 p.m., 2128 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

GASTROENTEROLOGY CONFERENCE
1st and 3rd Tuesday, 1 p.m., T-5 Sponsor VA Hospital Phoenix, Contact: Jasper L. McPhail, M.D. VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

GI CONFERENCE
(Special Program with U. of A. Consultants) 4th Friday, 1 p.m., T-5 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

G.I.-RADIOLOGY CLINICAL CORRELATION CONFERENCE
Each Tuesday, 1 p.m., T-5. Sponsor: VA Hospital Phoenix. Contact: Jasper L. McPhail, M.D., Veterans Administration Hospital. 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL SURGICAL G.I. CONFERENCE
1st & 3rd Friday - 2 p.m. -2128 Sponsor: VA Hospital, Phoenix. Contact: Jasper L. McPhail, M.D., VA Hospital, 7th Ave. & Indian School Rd., Phoenix. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

MEDICAL SERVICE GRAND ROUNDS
Each Friday, 11 a.m., T-5 Sponsor VA Hospital Phoenix Contact. Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education

SURGERY-PATHOLOGY CONFERENCE
Each Thursday, 7 a.m., 2128 Sponsor VA Hospital Phoenix Contact. Jasper L. McPhail, M.D., VA Hospital, 7th & Indian School Rd., Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

UROLOGY-PATHOLOGY CONFERENCE
Each Wednesday, 7 a.m., 2128 Sponsor VA Hospital Phoenix Contact Jasper L. McPhail, M.D., VA Hospital, 7th and Indian School Rd. Phoenix, AZ. Approved for required hours toward the ArMA Certificate in Continuing Medical Education.

Librax®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium Br.

Please consult complete prescribing information, a summary of which follows:

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

Final classification of the less-than-effective indications requires further investigation

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction, hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, i.e., dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

ROCHE

Roche Products Inc.
Manati, Puerto Rico 00701



Often a significant feature of irritable bowel syndrome*

The action of Librium®
(chlordiazepoxide HCl)

A significant advantage of adjunctive

Librax®

Each capsule contains
5 mg chlordiazepoxide HCl and
2.5 mg clidinium Br.

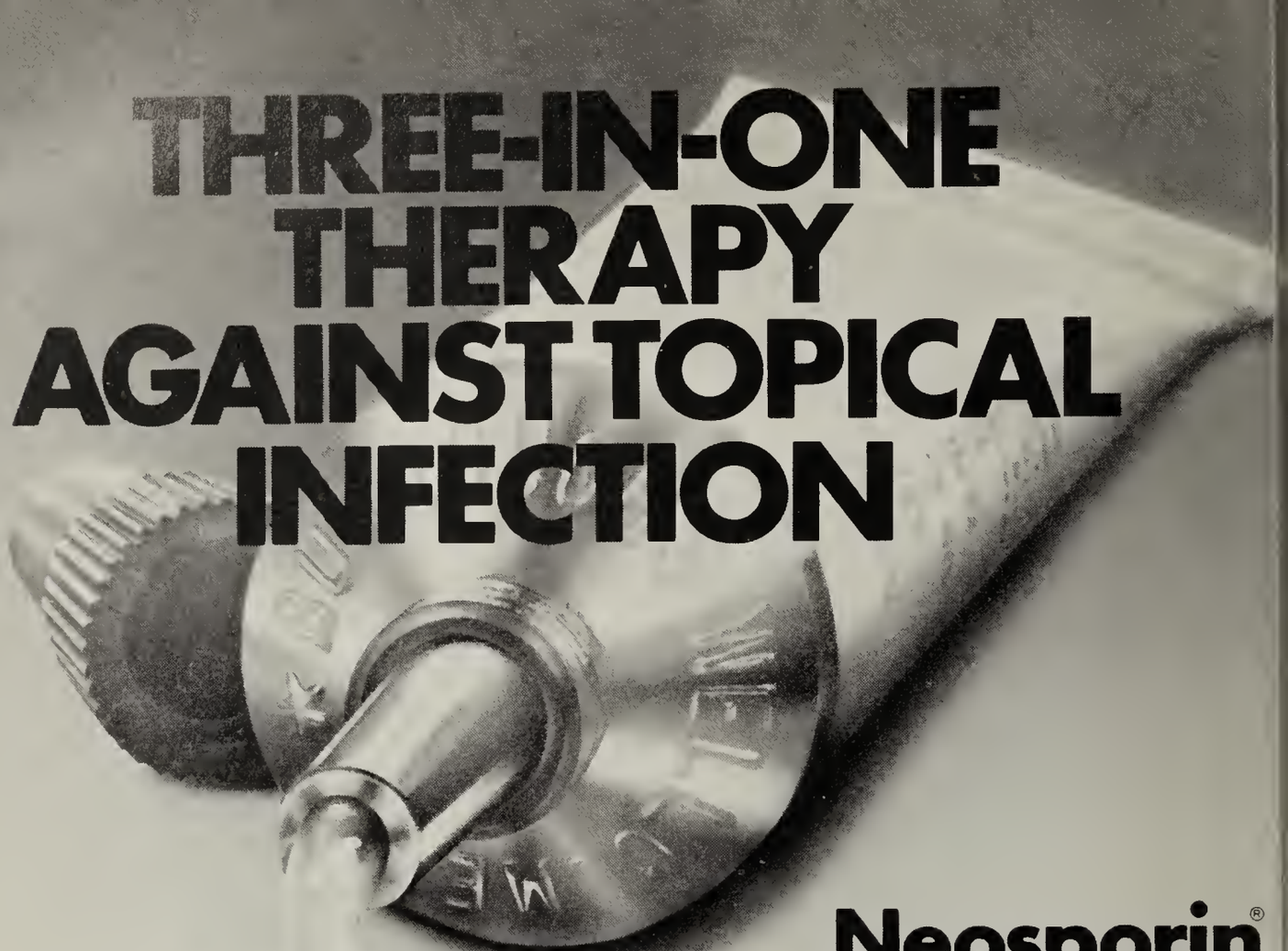
Antianxiety
Antisecretory
Antispasmodic

Librax is unique among G I. medications in providing the specific antianxiety action of Librium® (chlordiazepoxide HCl) as well as the potent antisecretory and antispasmodic actions of Quarzan® (clidinium Br) for adjunctive therapy of irritable bowel syndrome* and duodenal ulcer *

*Librax has been evaluated as possibly effective for this indication.
Please see brief summary of prescribing information on preceding page.

ROCHE

THREE-IN-ONE THERAPY AGAINST TOPICAL INFECTION



Neosporin® Ointment

(Polymyxin B-Bacitracin-Neomycin)

This potent broad-spectrum antibacterial provides overlapping action to help combat infection caused by common susceptible pathogens (including staph and strep). The petrolatum base is gently occlusive, protective and enhances spreading.

Neomycin

Staphylococcus
Haemophilus
Klebsiella
Aerobacter
Escherichia
Proteus
Corynebacterium
Streptococcus
Pneumococcus

Bacitracin

Staphylococcus
Corynebacterium
Streptococcus
Pneumococcus

Polymyxin B

Pseudomonas
Haemophilus
Klebsiella
Aerobacter
Escherichia

In vitro overlapping antibacterial action of Neosporin® Ointment (polymyxin B-bacitracin-neomycin).

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

Neosporin® Ointment

(Polymyxin B-Bacitracin-Neomycin)

Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 units; zinc bacitracin 400 units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

TRIAMTERENE CONSERVES POTASSIUM WHILE HYDROCHLOROTHIAZIDE LOWERS BLOOD PRESSURE

DYAZIDE®

Each capsule contains 50 mg. of Dyrenium® (triamterene, SK&F Co.) and 25 mg. of hydrochlorothiazide.

MAKES SENSE



Before preseribing, see eomplete preseribing information in SK&F Co. literature or PDR. A brief summary follows:

* Warning
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Assoeiated widened QRS eomplex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reaetions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

Supplied: Bottles of 100 and 1000 capsules; Single Unit Packages of 100 (intended for institutional use only).

FOR LONG-TERM CONTROL OF HYPERTENSION.* SERUM K^+ AND BUN SHOULD BE CHECKED PERIODICALLY. (SEE WARNINGS SECTION.)

SK&F CO., Carolina, P.R. 00630

SK&F CO.
a SmithKline company

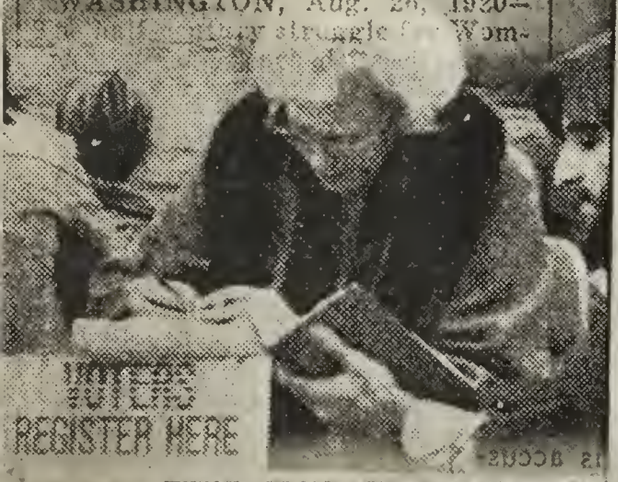
COLBY PROCLAIMS WOMAN SUFFRAGE

Signs Certificate of Ratification at His Home Without Women Witnesses.

MILITANTS VEXED AT PRIVACY.

Wanted Movies of Ceremony, But Both Factions Are

WASHINGTON, Aug. 26, 1920—[illegible] struggle [illegible] Wom-



Social Security Bill Is Signed; Gives Pensions to Aged, Jobl

Roosevelt Approves Message Intended to Benefit 30,000 Persons When States Adopt Cooperating Laws–He C the Measure 'Cornerstone' of His Economic Progra

WASHINGTON, Aug. 14, The Social Security Bill, pro a broad program of unemplo insurance and old age per and counted upon to benefit 20,000,000 persons, became l day when it was signed by dent Roosevelt in the prese those chiefly responsible fo ting it through C[illegible].

Mr. Roosevelt cal[illegible] me "the cor[illegible]erstone [illegible] str which is being [illegible] t me[illegible]s comple[illegible] ri[illegible]to [illegible]

COM

SENATE APPROVES 18-YEAR OLD VOTE IN ALL ELECTIONS

Amendment to Constitution is Sent to House, Where Passage is Expected

WASHINGTON, March 10, 1971—The Senate approved today, 94 to 0, and sent to [illegible]

JUL SIGNE the Draf Ends Nov

TRUMAN CLOSES ITED NATIONS CONFEREN WITH PLEA TO TRANSLATE CHARTER INTO DEEDS

NEW WORLD HOPE

President Hails 'Great Instrument of Peace,' Insists It Be Used

HISTORIC LANDMARK

Meeting Gives Standing Ovation as Executive Pictures Peace Gain

"If we fail to use it," he declared to the solemn final meeting of the delegates, 'we shall betray all of those who have died in order that we might meet here in freedom and safety to create it.'

"If we seek to use it selfishly—for the advantage of any one nation or any small group of nations—we shall be equally guilty of that betrayal."

Fervent Interpolation

The President, speaking in the auditorium of the War Memorial Opera House, built in memory of sons of the Golden Gate city who gave their lives in the first World War, in which he himself served, seemed to give unconscious expression to the solemn feeling of the occasion when, at the outset of his speech, he interpolated the words, half a hope, half a prayer:

"Oh, what a great day this can be in history!"

Just before the plenary session the President accompanied the

WASHINGTON, Jan. 27, 1973—"With the signing of the peace agreement in Paris today, and after receiving a report from the Secretary of the Army that



PATIENT PACKAGE INSERTS: A CONCEPT WHOSE TIME HAS COME?

The consumer's right to know is an irreversible and desirable trend of the Seventies. It extends, and properly, to a patient's right to know more about his or her prescription medications. One way, gaining favor, is through patient package inserts. Wisely-prepared and properly distributed when medically indicated, they could markedly improve patient knowledge and drug therapy—laudable goals by anyone's standards.

The PMA endorses these goals and will work with government, the health professions and consumers to achieve them.

The Advantages

The concept holds promise of benefits: better patient understanding of the product prescribed, better adherence to the treatment plan, and more awareness of possible side reactions.

Every doctor has had patients who fail to finish antibiotic regimens because they feel better. Some patients assume that if one tranquilizer or analgesic is good, two may be twice as good. Still others fail to report dizziness while on antihypertensive therapy —and so on.

Problems like these might arise less often if the patient received written information in addition to verbal instructions. Some studies suggest that patients are more receptive to such materials, and they more often understand the verbal instructions and follow them, when inserts are used.

The Disadvantages

There are also some potential problems. Obviously, the inserts must be clearly phrased, without extraneous or complex detail. How much information is enough? How can it be kept current? Should all patients receive the same information? Should inserts be included with all drugs? Should only potential problems be listed or are patients better off with a "fair balance" presentation that describes usefulness as well as drawbacks?

These and similar questions require answers, since model inserts have yet to be properly developed and tested. Despite the need for these studies, the FDA is proceeding prematurely with inserts on selected products. We think the Congress is the only place where the matter can be given the proper legal status and direction, particularly since it represents a conceptual change in the legal, medical and social framework of the nation's prescription drug information system.

The Solution

The PMA believes that carefully-devised pilot studies of various kinds of inserts are needed. They should be developed and implemented with full participation by doctors, pharmacists, consumers, communications experts and the drug industry. Such studies will provide reliable pathways to follow, so that inserts will be useful aids to medical practice.

And particularly we think that you should be closely involved in this debate and in these studies and decisions. Otherwise, people with less experience and qualifications may control the purposes, content and use of a tool with considerable promise for improved patient care. It could make a difference in your practice tomorrow, and more importantly, in the health of your patients.

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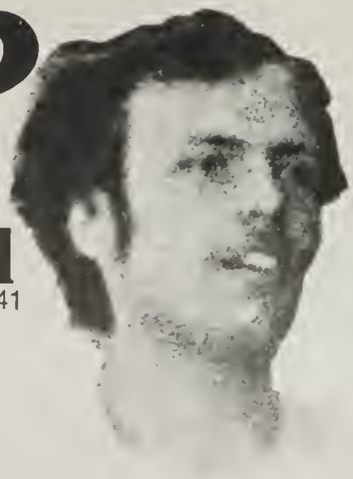
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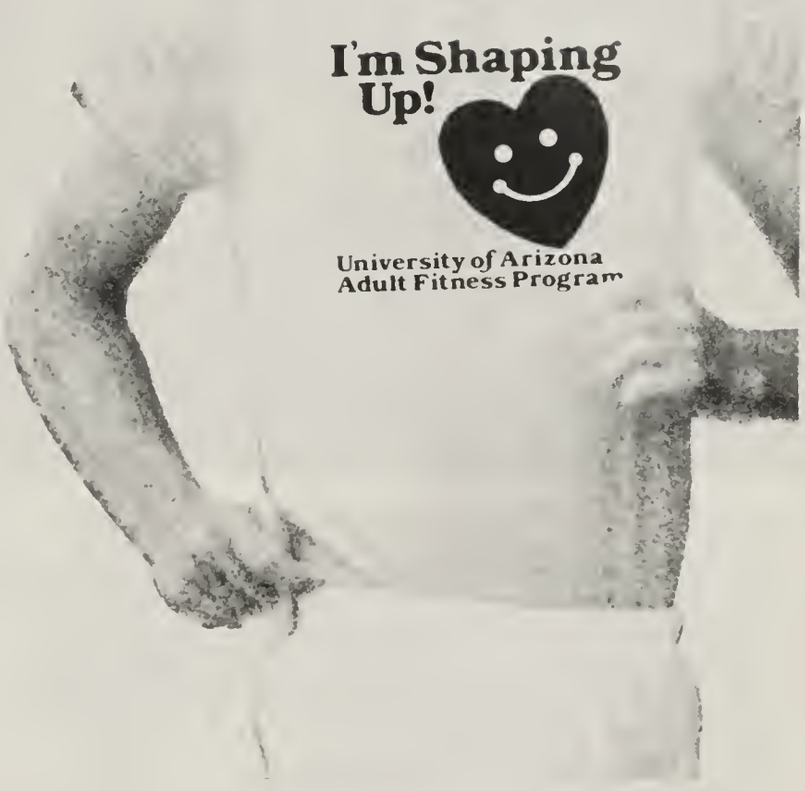


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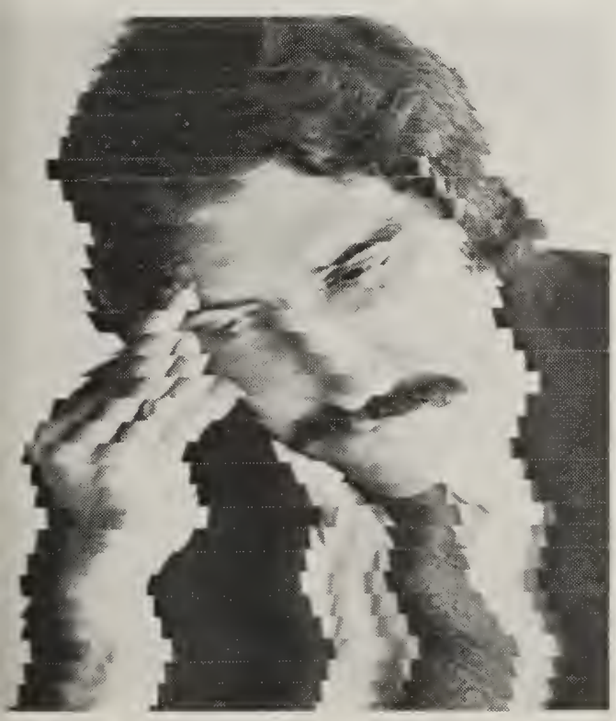
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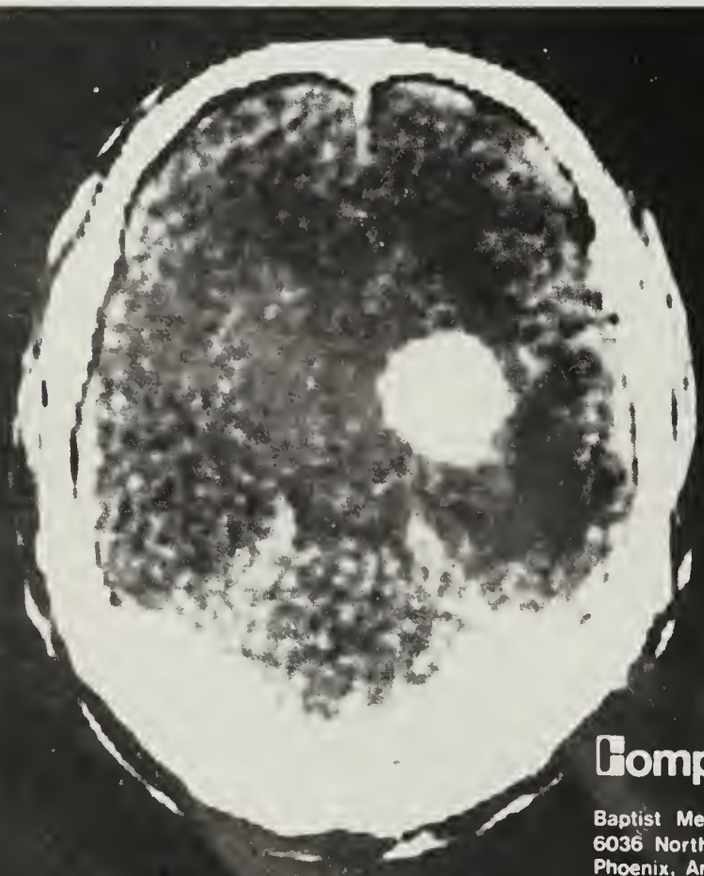
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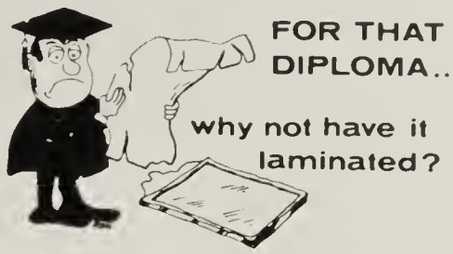
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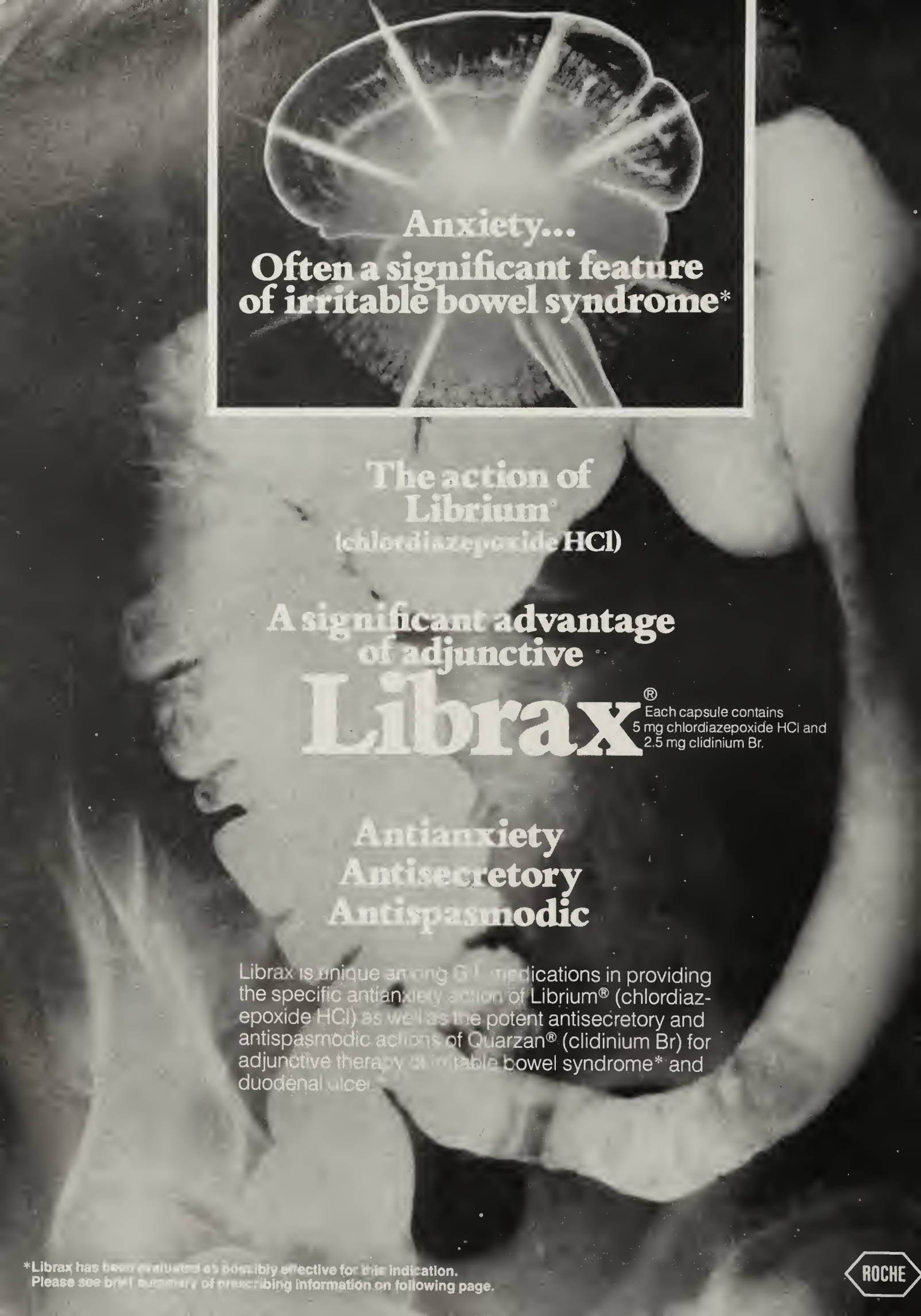
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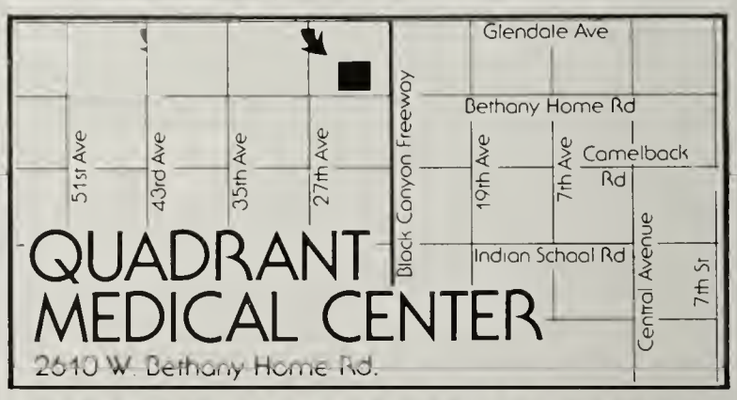
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Brief Summary of Prescribing Information
Combined TEGOPEN® (cloxacillin sodium)
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For complete information, consult Official Package Circular. (12) TEGOPEN 9/11/75

Indications: Although the principal indication for cloxacillin sodium is in the treatment of infections due to penicillinase-producing staphylococci, it may be used to initiate therapy in such patients in whom a staphylococcal infection is suspected. (See Important Note below.)

Bacteriologic studies to determine the causative organisms and their sensitivity to cloxacillin sodium should be performed.

Important Note: When it is judged necessary that treatment be initiated before definitive culture and sensitivity results are known, the choice of cloxacillin sodium should take into consideration the fact that it has been shown to be effective only in the treatment of infections caused by pneumococci, Group A beta-hemolytic streptococci, and penicillin G-resistant and penicillin G-sensitive staphylococci. If the bacteriology report later indicates the infection is due to an organism other than a penicillin G-resistant staphylococcus sensitive to cloxacillin sodium, the physician is advised to continue therapy with a drug other than cloxacillin sodium or any other penicillinase-resistant semi-synthetic penicillin.

Recent studies have reported that the percentage of staphylococcal isolates resistant to penicillin G outside the hospital is increasing, approximating the high percentage of resistant staphylococcal isolates found in the hospital. For this reason, it is recommended that a penicillinase-resistant penicillin be used as initial therapy for any suspected staphylococcal infection until culture and sensitivity results are known.

Cloxacillin sodium is a compound that acts through a mechanism similar to that of methicillin against penicillin G-resistant staphylococci. Strains of staphylococci resistant to methicillin have existed in nature and it is known that the number of these strains reported has been increasing. Such strains of staphylococci have been capable of producing serious disease, in some instances resulting in fatality. Because of this, there is concern that widespread use of the penicillinase-resistant penicillins may result in the appearance of an increasing number of staphylococcal strains which are resistant to these penicillins.

Methicillin-resistant strains are almost always resistant to all other penicillinase-resistant penicillins (cross-resistance with cephalosporin derivatives also occurs frequently). Resistance to any penicillinase-resistant penicillin should be interpreted as evidence of clinical resistance to all, in spite of the fact that minor variations in *in vitro* sensitivity may be encountered when more than one penicillinase-resistant penicillin is tested against the same strain of staphylococcus.

Contraindications: A history of a previous hypersensitivity reaction to any of the penicillins is a contraindication.

Warning: Serious and occasionally fatal hypersensitivity (anaphylactoid) reactions have been reported in patients on penicillin therapy. Although anaphylaxis is more frequent following parenteral therapy it has occurred in patients on oral penicillins. These reactions are more apt to occur in individuals with a history of sensitivity to multiple allergens.

There have been well documented reports of individuals with a history of penicillin hypersensitivity reactions who have experienced severe hypersensitivity reactions when treated with a cephalosporin. Before therapy with a penicillin, careful inquiry should be made concerning previous hypersensitivity reactions to penicillins, cephalosporins, and other allergens. If an allergic reaction occurs, the drug should be discontinued and the patient treated with the usual agents, e.g., pressor amines, antihistamines, and corticosteroids.

Safety for use in pregnancy has not been established.

Precautions: The possibility of the occurrence of superinfections with mycotic organisms or other pathogens should be kept in mind when using this compound, as with other antibiotics. If superinfection occurs during therapy, appropriate measures should be taken.

As with any potent drug, periodic assessment of organ system function, including renal, hepatic, and hematopoietic, should be made during long-term therapy.

Adverse Reactions: Gastrointestinal disturbances, such as nausea, epigastric discomfort, flatulence, and loose stools, have been noted by some patients. Mildly elevated SGOT levels (less than 100 units) have been reported in a few patients for whom pretherapeutic determinations were not made. Skin rashes and allergic symptoms, including wheezing and sneezing, have occasionally been encountered. Eosinophilia, with or without overt allergic manifestations, has been noted in some patients during therapy.

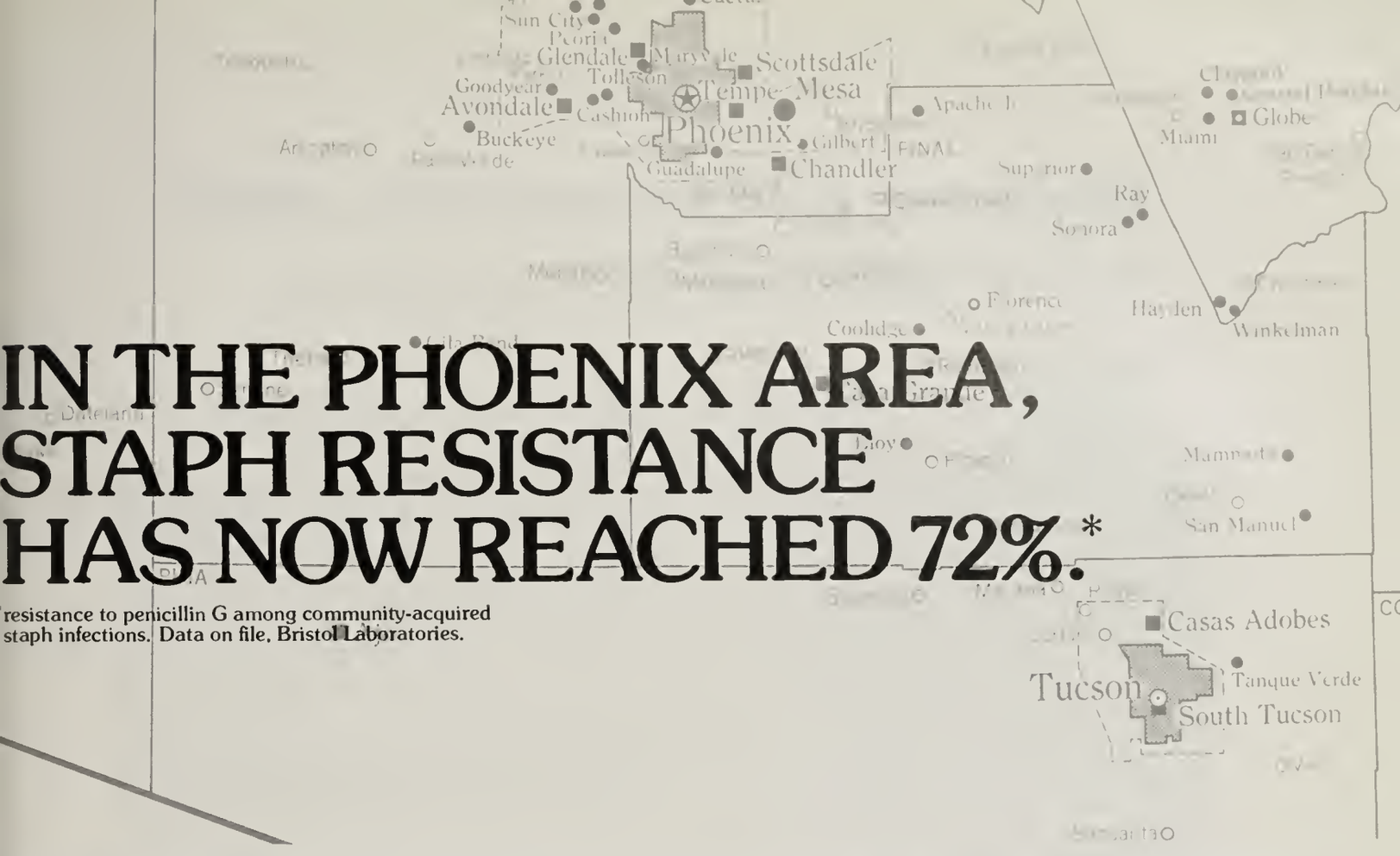
Usual Dosage: Adults: 250 mg. q.6h.

Children: 50 mg./Kg./day in equally divided doses q.6h. Children weighing more than 20 Kg. should be given the adult dose. Administer on empty stomach for maximum absorption.

N.B.: INFECTIONS CAUSED BY GROUP A BETA-HEMOLYTIC STREPTOCOCCI SHOULD BE TREATED FOR AT LEAST 10 DAYS TO HELP PREVENT THE OCCURRENCE OF ACUTE RHEUMATIC FEVER OR ACUTE GLOMERULONEPHRITIS.

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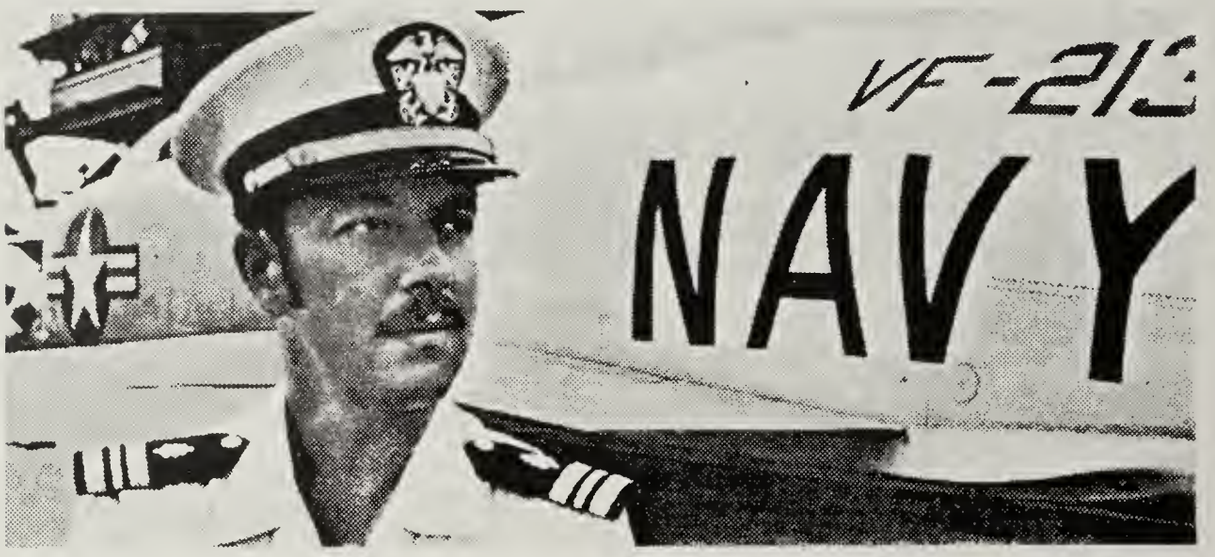
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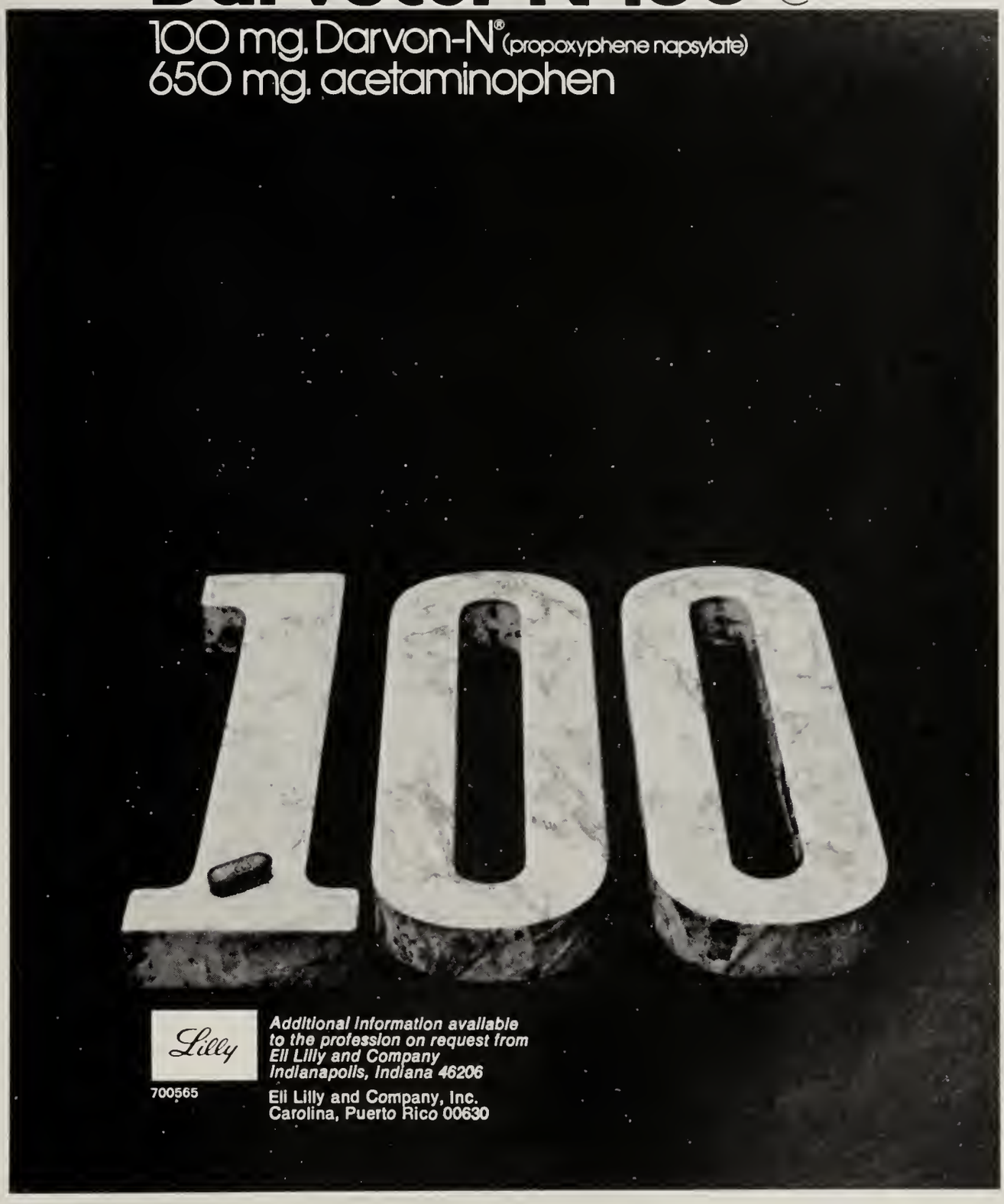
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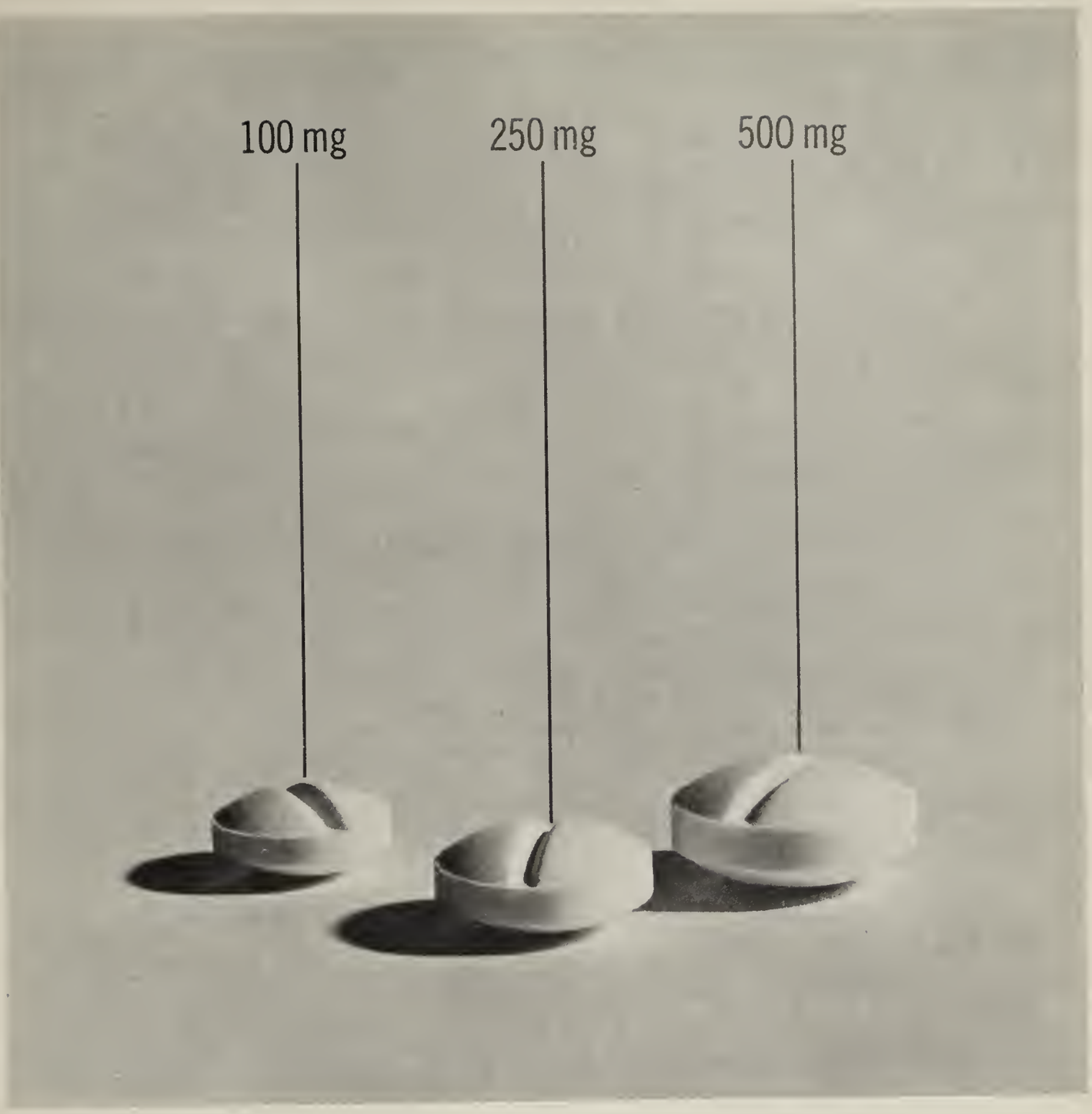
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Open to all practicing, licensed physicians in Arizona. Entries must be postmarked no later than June 15, 1978 and will become the property of ACP in Arizona. Judges will be Dr. Charles L. Krone, F.A.C.P., Dr. A.C. van Ravenswaay, F.A.C.P., and Dr. Ashton B. Taylor, F.A.C.P. All decisions will be final. Winner will be announced in August issue of Arizona Medicine.

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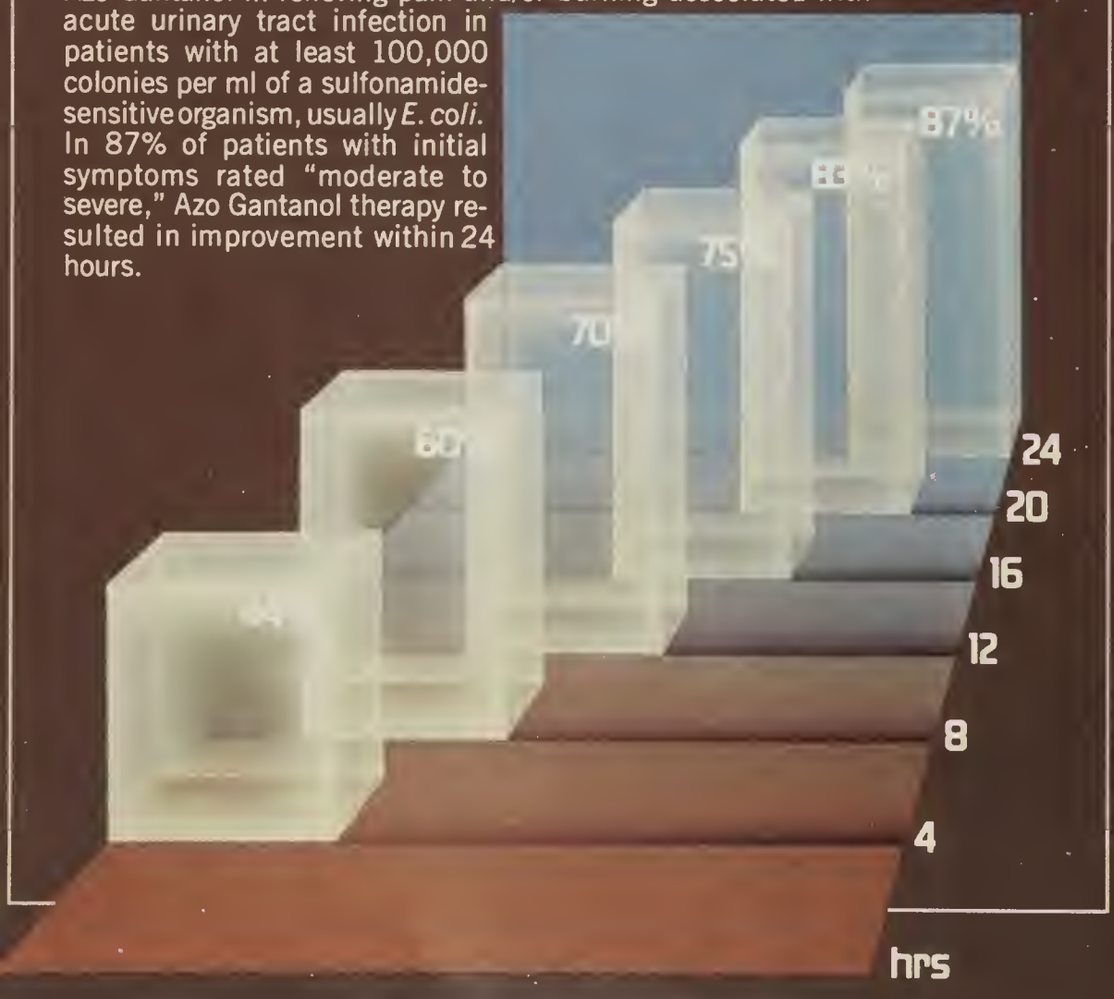
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Indications: In adults, urinary tract infections complicated by pain (primarily pyelonephritis, pyelitis and cystitis) due to susceptible organis (usually *E. coli, Klebsiella-Aerobacter, Staphylo coccus aureus, Proteus mirabilis*, and, less fre quently, *Proteus vulgaris*) in the absence of obstructive uropathy or foreign bodies. **Note: Ca fully coordinate *in vitro* sulfonamide sensitivity tests with bacteriologic and clinical response; a aminobenzoic acid to follow-up culture media. increasing frequency of resistant organisms lim the usefulness of antibacterials including sul fonamides.** Measure sulfonamide blood levels a variations may occur; 20 mg/100 ml should be maximum total level.

Contraindications: Children below age 12; sul fonamide hypersensitivity; pregnancy at term an during nursing period; because Azo Gantanol co tains phenazopyridine hydrochloride it is contra dicated in glomerulonephritis, severe hepatitis, uremia, and pyelonephritis of pregnancy with G disturbances.

Warnings: Safety during pregnancy not establish Deaths from hypersensitivity reactions, agranulo tosis, aplastic anemia and other blood dyscrasias have been reported and early clinical signs (sore throat, fever, pallor, purpura or jaundice) may in dicate serious blood disorders. Frequent CBC an urinalysis with microscopic examination are rec ommended during sulfonamide therapy.

Precautions: Use cautiously in patients with im paired renal or hepatic function, severe allergy, bronchial asthma; in glucose-6-phosphate dehydrogenase-deficient individuals in whom dose-related hemolysis may occur. Maintain adequate fluid intake to prevent crystalluria and stone formation.

Adverse Reactions: *Blood dyscrasias* (agran ulocytosis, aplastic anemia, thrombocytopenia, leukopenia, hemolytic anemia, purpura, hypopr thrombinemia and methemoglobinemia); *allergi reactions* (erythema multiforme, skin eruptions, Stevens-Johnson syndrome, epidermal necrolysis urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photo sensitization, arthralgia and allergic myocarditis); *G.I. reactions* (nausea, emesis, abdominal pains hepatitis, diarrhea, anorexia, pancreatitis and stomatitis); *CNS reactions* (headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo and insomnia); *miscellaneous reactions* (drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon). Due to certain chemical similarities with some goitrogens, di uretics (acetazolamide, thiazides) and oral hypo glycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypo glycemia. Cross-sensitivity with these agents may exist.

Dosage: Azo Gantanol is intended for the acute, painful phase of urinary tract infections. *Usual adult dosage:* 2 Gm (4 tabs) initially, then 1 Gm (2 tabs) B.I.D. for up to 3 days. If pain persists, causes other than infection should be sought. After relief of pain has been obtained, continued treatment with Gantanol (sulfamethoxazole) may be considered.

NOTE: Patients should be told that the orange-r dye (phenazopyridine HCl) will color the urine.

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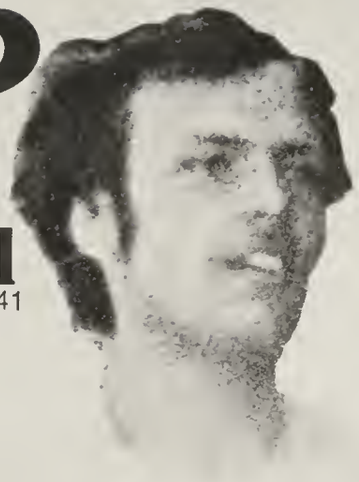
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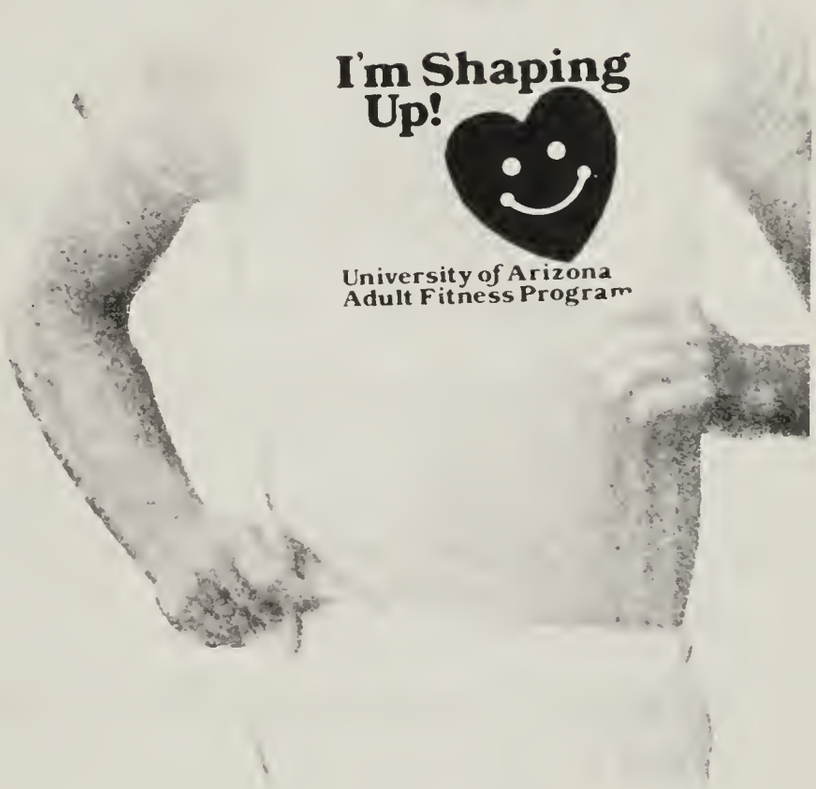


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The Challenges Of The Future

The College of Medicine is pleased to assist in the preparation of the Third Annual issue of ARIZONA MEDICINE by providing articles developed by our faculty. The theme of this year's university edition focuses on diagnostic advances in clinical medicine. The articles are written for the practicing physician. They tend to be synoptic and, where appropriate, review developments that are now available.

Today the challenge of the academic health center is to develop, assess, and apply new techniques and strategies for the diagnosis and treatment of disease. The challenge of the future will be to integrate the expanding role of technology with our current levels of investigation.

As we identify new techniques, old techniques will be discarded or refined and strengthened. The cost, effectiveness, and benefits of new diagnostic strategies must be carefully weighed to ensure that our patients will continue to profit from these strategies.

Anthony F. Vutoro, M.D.

Associate Dean
Office of Continuing Medical Education
and Outreach

An Introduction To B-Scan Ultrasonography

For The Primary Care Physician

Kai Haber, M.D.

Man is the latest animal to utilize high frequency sound waves in attempting to solve biological problems. For millions of years, bats have been using ultrasonics for orientation and food-finding purposes in the air, while porpoises and dolphins have evolved similar abilities for use in the aquatic environment.

Ultrasound encompasses that range of the sonic energy spectrum above the level of human hearing (namely approximately 15,000 to 20,000 cycles per second). Virtually all of the diagnostic medical applications of ultrasound involve ultrasonic frequencies in the range between 2 and 10 million cycles per second (2-10 megahertz). Most diagnostic ultrasonic instrumentation utilizes pulse-echo ranging technique. This technique is analogous to locating objects under water by Sonar. Unlike x-rays and gamma rays, ultrasonic waves, being accoustical energy, require a medium for propagation. The denser the medium through which the ultrasonic wave is traveling, the greater the velocity. A short burst of ultrasonic energy which is transmitted into a homogeneous medium will travel in a forward direction until the energy is dissipated within that medium by absorption. However, if the sonic pulse encounters a second medium which is of sufficiently different density from the first medium, a portion of that pulse will be reflected at the interface between the media. The remainder of the pulse will continue on in a relatively linear fashion. It is this reflected portion of the pulse, the echo, which is the signal used in generating an ultrasonic image. The strength of the returning echo will vary according not only to the distance that the reflecting surface is from the receiver but also on the strength of the reflecting surface, that is to say the density difference of the two media forming the reflective interface. The greater this difference, the stronger the returning echo at any given depth. The body contains many acoustic interfaces. By varying the direction of the ultrasonic beam a composite of the reflections can be generated and stored electronically to produce a two dimensional image.

The ultrasonic pulse is generated by an ultrasonic transducer. This consists of a crystal which oscillates at a very high frequency when an electrical impulse is imparted to it. These mechanical oscillations form the ultrasonic wave. Conversely, the same transducer will generate a small electrical impulse when the transducer

From: University of Arizona, Health Sciences Center, Tucson, AZ 85724, Dr. Haber, Assit. Professor of Radiology, Picker Scholar, James Picker Foundation.



FIGURE 1
Fetal detail with gray-scale ultrasound has become exquisite.

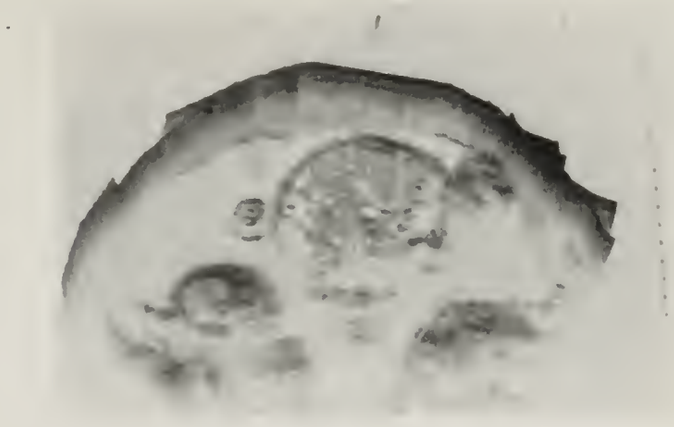


FIGURE 2
Excellent sonographic fetal detail in normal pregnancy is due to a large degree to the transonic nature of amniotic fluid.

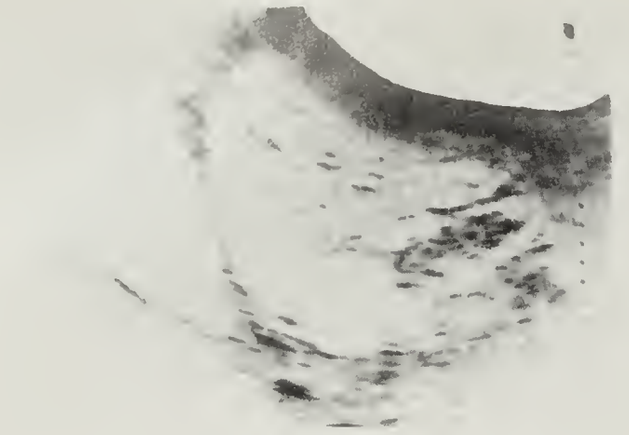
element is stimulated by high frequency oscillations (ultrasound). Thus, the transducer acts not only as a transmitter, but also as a receiver.

Most diagnostic ultrasonic instrumentation measures the time between transmission of the pulse and reception of an echo. Knowing the velocity of ultrasound in soft tissues, that time can be converted into a distance. In addition to measuring this time, the strength (amplitude) of the returning echo is also determined. These are the two basic forms of information which are then translated into an image. In addition, various strengths of echo can be assigned certain levels of brightness which will be displayed as certain shades of gray on the final photographic image. Thus, the term B-mode ultrasonic imaging is an abbreviation of "brightness-mode". The term "gray-scale" simply refers to the fact that these various brightnesses are perceived as a series of some 16 to 32 shades of gray.

The above description of the basic physics and instrumentation involved in B-mode ultrasonography is, of course, quite simplified. Those readers wishing slightly more depth are referred to the first three chapters in the excellent monography on gray-scale ultrasonography by Bartrum and Crow.[1] Although ultrasonics has been utilized in diagnostic applications since the early 1950's, it was not until the middle 1960's that this imaging modality came into widespread use. Within the last five years there have been dramatic advances in the quality of diagnostic ultrasonic instrumentation and this modality is now an integral part of diagnostic imaging in virtually every medical community.

There is now a large body of both clinical and basic research utilizing sonic waves of the same frequency and power as utilized in every day ultrasonic examinations. To date, there is no evidence that the frequencies and energies employed in diagnostic ultrasonography cause any biological effect, either harmful or beneficial.

Thus, ultrasound is certainly the superior imaging modality in obstetrics. In addition to there being no ionizing radiation to the mother or the fetus, the quality of the images are far superior to those obtained with standard radiographic techniques or even with contrast instilled in the amniotic cavity. It is considered routine to be able to visualize the five digits of the hand and to be able to visualize the intercostal spaces in the fetus. Exquisite intrathoracic and intra-abdominal detail is the rule, rather than the exception. (Figures 1 and 2). Various measurements of the fetus can be made *in utero*. The most common of these measurements is the bi-parietal diameter (BPD). By careful, serial measurements of the BPD during the course of the pregnancy, the gestational age can be determined with a high degree of confidence. Placental localization and detection of placental abnormalities, such as placenta abruptio and early degeneration of the placenta, can be readily detected. Ultrasonic imaging has become a routine early examination performed in the evaluation of third trimester bleeding. Placenta previa can be readily diagnosed. Polyhydramnios, oligohydramnios,



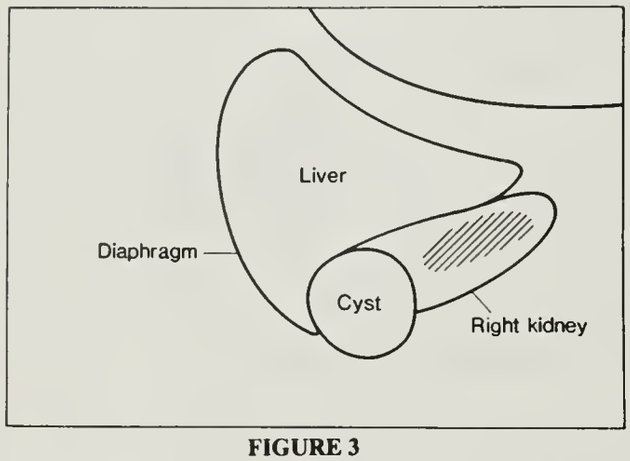
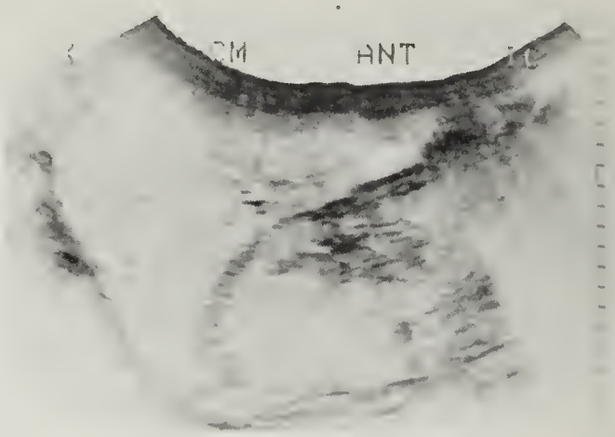


FIGURE 3
Longitudinal sonogram made 8 cm to the right of the patient's midline in the supine position. Note echo-free nature of the renal cyst.



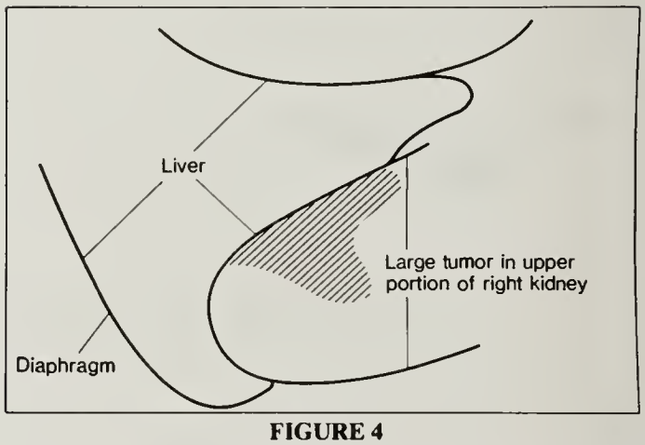


FIGURE 4
Longitudinal sonogram made 7 cm to the right of the midline, with patient supine. The normal architecture in the upper half of the kidney has been replaced by tumor. X marks xyphoid and CM marks lower costal margin.

fetal abnormalities, presence or absence of multiple gestations, and fetal demise are all diagnoses which can be made by ultrasonography with a high degree of accuracy.[2,3] It has now become routine at many community hospitals and in many office practices to determine a site for amniocentesis by ultrasonic imaging. The ultrasonic examination can give a rapid image showing where needle placement would be optimal without danger of the needle passing through the placenta or a fetal part.

Gray scale B-scanning has had a major impact in areas other than obstetrics. Its use in the upper abdomen has become widespread in the evaluation of various disease states. Its usefullness has been proven in virtually every organ system within the abdomen, retroperitoneum and pelvis. Space permits only a rather superficial overview of the major applications in these regions.

Certainly one of the most reliable and useful applications of ultrasound has been in distinguishing between the cystic or solid nature of a mass. This, of course, has a prime application in the evaluation of renal masses, as cystic renal masses have a very high probability of being benign. Since cystic from solid renal masses can not be reliably distinguished from one another by intravenous urography, ultrasonography is indicated if a mass is discovered on a urogram. (Figures 3 and 4).

In addition to imaging a renal cyst, its depth can accurately by measured from the ultrasonic scan. This three dimensional information allows rapid and accurate percutaneous aspiration of a renal cyst.

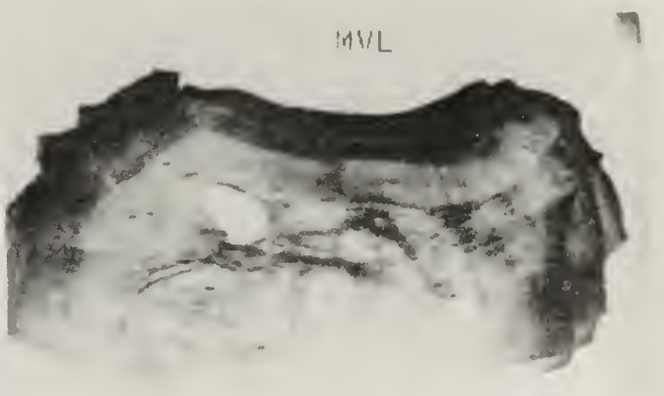
Since intravenous urography and radionuclide renal imaging both depend upon at least a minimal degree of renal function, ultrasonography is a very useful modality in defining renal morphology in cases of either unilateral or bilateral renal failure. In addition to imaging the kidneys, the perinephric regions are readily visualized. Retroperitoneal tumors, abscesses and hemorrhage can all be imaged by ultrasound.

Increased resolution which is available with the latest generation of gray-scale ultrasonic B-scanners allows imaging of the liver in great detail. Abnormalities of the hepatic or portal venous systems and of the biliary system can be visualized. In many instances the medical and surgical types of jaundice can be differentiated from one another. Frequently, calculi in the common bile duct, masses in the porta hepatis or a mass in the head of the pancreas can be imaged in addition to the distended common bile duct and intrahepatic biliary radicles in cases of surgical jaundice. Solid masses (e.g. metastatic foci) of only several milimeters in diameter can be recognized.

Ultrasonography is very helpful in the evaluation of cholelithiasis. (Figure 5). In many institutions, ultrasonography is the initial step in the work up of suspected gallstones. In others, ultrasonography is performed when there is non-visualization on an oral cholecystogram or when the oral cholecystogram is equivocal. Certainly,



FIGURE 5
Sonogram of gallbaldder with multiple gallstones. Note acoustic shadow cast by the largest gallstone.



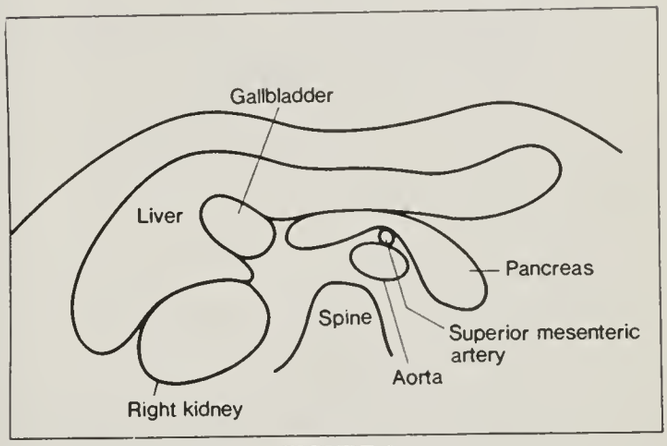


FIGURE 6
Supine transverse sonogram of the upper abdomen showing relationship of the pancreas to neighboring structures.

ultrasonography is a valuable diagnostic modality in evaluation of the patient suspected of gallstones. Visualization of the gallbladder by ultrasonography is not dependent upon function.

The pancreas can be visualized in a high percentage of patients (Figure 6).[4] However, in some there is a significant amount of gas between the surface of the abdomen and pancreas. Gas is a total reflector of ultrasound and a significant collection of gas cannot be penetrated by the ultrasonic beam. Thus, the pancreas is not always imaged by ultrasonography. This appears to be the major drawback of ultrasonic evaluation of the pancreas. Such entities as carcinoma of the pancreas, pancreatitis and pancreatic pseudocyst can be imaged and diagnosed. However, there is overlap between the ultrasonic image of carcinoma of the pancreas and chronic pancreatitis. Thus, the distinction between the two is not sharply defined. Correlation with clinical data and other diagnostic findings is a very important adjunct to the ultrasonic examination. Ultrasonography is the imaging modality of choice in the evaluation of a patient suspected of pancreatic pseudocyst or in follow-up examinations of pancreatic pseudocyst or pancreatic carcinoma.

Ultrasound has proven to be the modality of choice for evaluation of abdominal aortic aneurysm. Aneurysm size can be measured quite accurately. The extent of the aneurysm can be visualized and the amount of clot or atheromatous change relative to the degree of patency can be determined (Figure 7). Furthermore, because of its benign nature, serial ultrasonography can be performed at frequent intervals in follow-up examinations in patients with borderline aneurysmal dilation of the aorta.

The presence or absence of abscess within the abdomen can frequently be determined on the basis of an ultrasonic examination. This is often the case in spite of a multitude of negative standard radiographic procedures. The major problem in the diagnosis of intraabdominal or intraperitoneal abscess detection is interference of the sonic beam by bowel gas, as these patients frequently have small bowel ileus.

The spleen can be readily visualized and a multitude of splenic abnormalities can be detected.[5] Diaphragmatic motion, particularly on the right side, can be accurately measured and permanently recorded with this modality (Figure 8).[6] Visualization of the diaphragm on the left side is more difficult because of the presence of gas in the stomach and splenic flexure.

Diagnostic ultrasonography has great utility in evaluation of pelvic abnormalities. The uterus and ovaries can be routinely imaged, even in the normal state. A partial list of abnormalities which can be detected are as follows: prostatic enlargement, ovarian neoplasms and cysts, uterine myomas, abscesses, localized areas of acitic fluid or hemorrhage, and abnormalities of the bladder and lymphadenopathy.

Other areas in which gray scale ultrasonic B-scanning has proven to be of great clinical utility include the detection and localization of pleural fluid, the evaluation

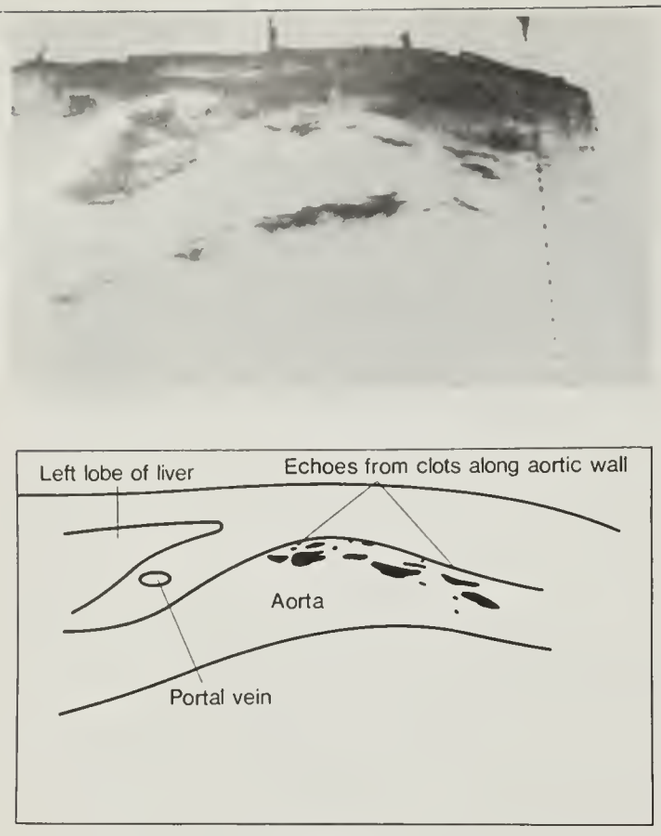


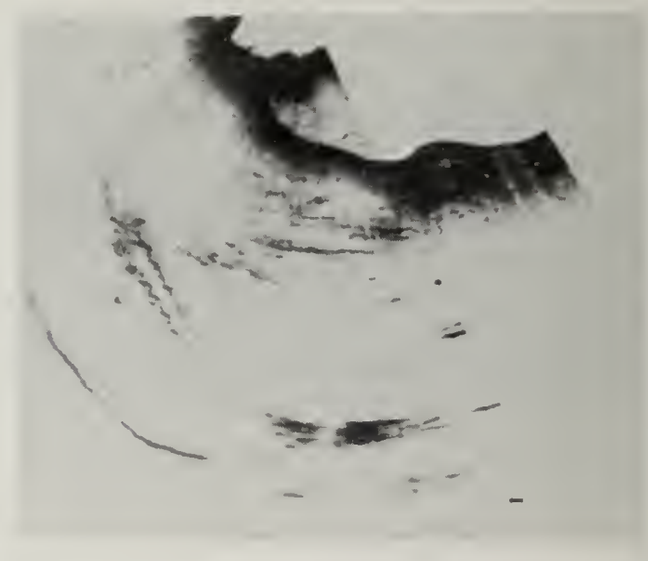
FIGURE 7

Aneurysm of abdominal aorta. Note echos from blood clots which form in areas of slowed blood flow. The small dots on right hand side of echogram are 1 cm apart. The aneurysm is 5.2 cm in diameter. The normal aortic lumen is 2 cm or less.

of thyroid masses, Baker's cyst, parotid abnormalities, testicular masses, and virtually any other soft tissue masses superficially located in other parts of the body.

Because of the non-invasive and benign nature of ultrasonic examinations, there has been great scientific and commercial impetus for development of newer and more refined ultrasonic instrumentation. Two particularly promising developments have been the application of real-time two-dimensional ultrasonic imaging and the development in Australia of an automated gray scale B-mode scanning system.[7] Real-time ultrasonic imaging refers to rapid collection of ultrasonic images ranging from one to sixty images per second. This allows visualization of motion within the body, which is particularly useful in detection of abnormalities of the heart and in rapidly determining the presence or absence of fetal life. Unfortunately, real-time imaging results in some degradation of the ultrasonic image because of the rapid acquisition and display of the data. However, in spite of this drawback, real-time ultrasonic imaging appears to have a very bright future. The recent development and commercial availability of the Octoson (from Australia) promises to make major strides in the development of standardization of ultrasonic images, analogous to the making of routine radiographs. In this system, the transducers are separated from the patient by a water bath. The transducers are manipulated by remote control and a high degree of image reproduceability is achieved.

Ultrasonography has advanced markedly within the last ten years and will continue to have a greater and greater impact upon diagnostic imaging in the future. For



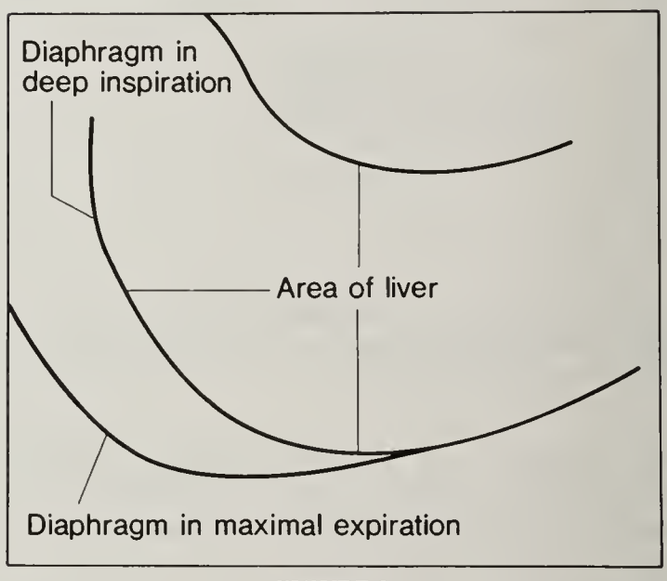


FIGURE 8

Longitudinal B-scan of the diaphragm showing diaphragmatic position at end of deep inspiration and end of complete expiration. The same area was, thus, scanned twice giving "compound" scan.

the most part, ultrasonic imaging is complimentary and not competitive with other imaging modalities such as routine radiography, computerized tomography and radionuclide imaging.

All figures and legends are from a chapter by the author in Imaging in Diagnostic Medicine, Sol Nudelman, editor. Plenum Press. In press.

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Does it influence your choice of a peripheral/cerebral vasodilator*?

- Vasodilan—compatible with coexisting diseases
- Vasodilan—compatible with concomitant therapy
- Vasodilan—compatible with your total regimen for vascular insufficiency

*Indications: Based on a review of this drug by the National Academy of Sciences-National Research Council and/or other information, the FDA has classified the indications as follows:

Possibly Effective:

1. For the relief of symptoms associated with cerebral vascular insufficiency.
2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.

Final classification of the less-than-effective indications requires further investigation.

Composition: Vasodilan tablets, isoxsuprine HCl, 10 mg. and 20 mg.

Vasodilan injection, isoxsuprine HCl, 5 mg., per ml.

Dosage and Administration: Oral: 10 to 20 mg., three or four times daily.

Intramuscular: 5 to 10 mg. (1 or 2 ml.) two or three times daily. Intramuscular administration may be used initially in severe or acute conditions.

Contraindications and Cautions: There are no known contraindications to oral use when administered in recommended doses. Should not be given immediately postpartum or in the presence of arterial bleeding.

Parenteral administration is not recommended in the presence of hypotension or tachycardia.

Intravenous administration should not be given because of increased likelihood of side effects.

Adverse Reactions: On rare occasions oral administration of the drug has been associated in time with the occurrence of hypotension, tachycardia, nausea, vomiting, dizziness, abdominal distress, and severe rash. If rash appears the drug should be discontinued.

Although available evidence suggests a temporal association of these reactions with isoxsuprine, a causal relationship can be neither confirmed nor refuted.

Administration of single dose of 10 mg. intramuscularly may result in hypotension and tachycardia. These symptoms are more pronounced in higher doses. For these reasons single intramuscular doses exceeding 10 mg. are not recommended. Repeated administration of 5 to 10 mg. intramuscularly at suitable intervals may be employed.

Supplied: Tablets, 10 mg., bottles of 100, 1000, 5000 and Unit Dose; Tablets, 20 mg., bottles of 100, 500, 1000, 5000 and Unit Dose, Injection, 10 mg. per 2 ml. ampul, box of six 2 ml. ampuls.

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VASODILAN®
(ISOXSUPRINE HCl)
20-mg tablets

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This asthmatic isn't worried about his



next breath...

he's active
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Each capsule ar tablespaonful (15 ml) elixir contains theaphylline (anhydraus) 150 mg and glyceryl guaiacalate (guaifenesin) 90 mg. Elixir: alcahal 15%

high theophylline for effective around-the-clock therapy

Quibron may give the asthmatic up to eight hours of bronchodilation with each dose and provides the high dosages of theophylline which are now believed necessary to keep patients free of acute attacks and chronic wheezing.

100% free theophylline

Quibron helps achieve high serum theophylline levels with minimal dosage volume...delivers 100% free theophylline in comparison to many other compounds which contain from 47% to 91% effective theophylline.

individualized theophylline dosage schedule

Today's more efficient usage of theophylline includes individualizing dosage and monitoring serum theophylline levels. The usual recommended dosages of Quibron are: Adults—1 to 2 capsules or tablespoonfuls every 6 to 8 hours; dosage may be cautiously adjusted upward when necessary to a maximum of 2000 mg theophylline per 24 hours. Children under 12—4 to 6 mg theophylline per kg/body weight every 6 to 8 hours; dosage may be cautiously adjusted up to 9 or 10 mg/kg every 6 hours.

Now, for the asthmatic who requires high-dose theophylline therapy for therapeutic serum concentrations

Mead Johnson Pharmaceutical Division announces

QUIBRON®-300

Each capsule contains 300 mg theophylline (anhydrous) and 180 mg glyceryl guaiacolate (guaifenesin)

For Brief Summary, please see the last page of this advertisement.

QUIBRON®-300

Each capsule contains 300 mg theophylline (anhydrous) and 180 mg glyceryl guaiacolate (guaifenesin)

The new high-dose theophylline capsule... for dependable theophylline therapy when products of lower dosage do not adequately control asthma symptoms.

Specially formulated

...for optimal efficacy

Quibron-300 is appropriate therapy for asthma patients whose symptoms are not adequately controlled on lower doses of theophylline, particularly for patients whose theophylline dosage has been adjusted upward to achieve therapeutic serum levels. In one study,[1] an average peak increase in FEV_1 of 35% was demonstrated after a single dose equivalent to one Quibron-300 capsule, and significant improvement in this pulmonary function lasted for nearly eight hours after administration.

...for optimal predictability

One Quibron-300 capsule q6-8h yields therapeutic serum levels (10-20 mcg/ml) in many adults. With a single dose, more than 75% of patients achieved serum levels potentially providing clinical benefit (5-15 mcg/ml). Half-life of theophylline varies widely from patient to patient, making monitoring of theophylline therapy important. Patient response may be monitored clinically if blood levels are not available as long as dosage does not exceed 1200 mg in 24 hours for adults.

...for optimal dosage convenience

The simple, convenient dosage of new Quibron-300—one capsule every six to eight hours—makes it easy for patients to comply with high-dose regimens often required to achieve therapeutic serum levels. Quibron-300 capsules may provide maximum therapeutic value with maximum convenience. In fact, the switch from a low-dose to a high-dose regimen may be accomplished by merely switching capsules, by stepping up to Quibron-300 capsules.

...for minimal theophylline side effects

Adverse reactions to theophylline are related to serum levels and are usually not a problem at concentrations below 20 mcg/ml. Of 45 patients studied[1] after a single dose, only seven reported adverse reactions. The most common reaction was a feeling of lightheadedness by three of these seven patients.

Reference 1. Data on file. Mead Johnson Pharmaceutical Division.

Indications: For the symptomatic treatment of bronchospastic conditions such as bronchial asthma, asthmatic bronchitis, chronic bronchitis, and pulmonary emphysema.

Dosage: Quibron—Adults: 1-2 capsules or 1-2 tablespoonfuls elixir every 6-8 hours. Children under 12: 4-6 mg theophylline/kg body weight every 6-8 hours.

Quibron-300— Adults: 1 capsule every 6-8 hours.

Theophylline dosage may be cautiously increased to 2000 mg/24 hour in adults and 9 or 10 mg/kg every 6 hours in children. Monitoring of serum theophylline levels at higher dosages is recommended.

Precautions: Do not administer more frequently than every 6 hours, or within 12 hours after rectal dose of any preparation containing theophylline or aminophylline. Do not give other xanthine derivatives concurrently. Use in case of pregnancy only when clearly needed.

Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea, and vomiting. The frequency of adverse reactions is related to the serum theophylline level and is not usually a problem at serum theophylline levels below 20 μg/ml.

How Supplied: Quibron Elixir: Bottles of 1 pint and 1 gallon. Quibron Capsules: Bottles of 100 and 1000 and unit-dose packs of 100. Quibron-300 Capsules: Bottles of 100.

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MJL 7-4280

Advances In Cardiovascular Nuclear Medicine

Mark J. Friedman, M.D.
Robert D. Okada, M.D.
Steven Goldman, M.D.

Nuclear medicine techniques have recently been added to the practicing physician's armamentarium for the diagnosis and evaluation of cardiovascular disease. The development of new radionuclides and the introduction of advanced types of scintillation cameras and sophisticated computer techniques for data analysis allow the acquisition of information which is frequently not obtainable by more conventional procedures. In addition, most of the nuclear cardiology studies are performed in a relatively non-invasive fashion requiring only an intravenous injection of the radionuclide and can be repeated serially without significant morbidity or patient inconvenience.

At the present time, cardiovascular nuclear medicine procedures can be used to assess the presence of myocardial ischemia and infarction as well as to assess cardiac function. Radionuclides such as technetium-99m pyrophosphate (Tc-99m PYP) accumulate maximally in zones of myocardial infarction or necrosis, while other agents such as thallium-201 accumulate maximally in normally perfused myocardium. Thus, these two radionuclides can be used to assess myocardial perfusion and viability. Radionuclides which remain within the intravascular space during the study period are utilized for the evaluation of ventricular ejection fraction and regional ventricular wall motion.

We will review the commonly available cardiovascular radionuclide techniques and discuss recommendations for their use.

MYOCARDIAL INFARCTION SCINTIGRAPHY

Technetium-99m pyrophosphate is a radionuclide which accumulates maximally in zones of myocardial infarction to produce an area of increased radioactivity (Figs. 1B & 1C). The radionuclide is injected into a peripheral vein and cardiac imaging can be performed 1-3 hours after the injection. The pyrophosphate scan may be positive as early as 10-12 hours after the onset of myocardial infarction, and zones of increased radioactivity are usually found 24-48 hours after infarction.

From: The Department of Internal Medicine, Section of Cardiology, Tucson Veterans Administration Hospital and the University of Arizona Health Sciences Center, Tucson, AZ. Requests for reprints to M.J. Friedman, Section of Cardiology, Tucson Veterans Administration Hospital, South 6th Avenue, Tucson, AZ 85723.

Repeat injection of Tc-99m PYP and scanning 4-6 days after acute myocardial infarction may demonstrate a persistently positive scan, however, a negative scan is frequently noted by the 14th post-infarction day.[1]

In a recent study reported from the University of Arizona Health Sciences Center,[2] 101 patients admitted to the Coronary Care Unit for evaluation of myocardial infarction had Tc-99m PYP scans performed within 72 hours of admission. The Tc-99m PYP scan was positive in 21/21 patients who had definite transmural myocardial infarction and 7/7 patients who had definite non-transmural myocardial infarction. The scans frequently localized the myocardial infarction to the anterior or inferior surface of the heart (Figs. 1B & 1C.). This localization correlated well with electrocardiographic localization of the myocardial infarction. This high degree of sensitivity agrees with most previously reported studies for acute transmural myocardial infarction.[1,3] However, a recent report[4] has questioned the sensitivity of the Tc-99m PYP scan in patients with acute transmural myocardial infarction and others[5] have found the Tc-99m PYP scan to be less sensitive for acute non-transmural myocardial infarction. In addition, 52/52 patients without evidence of acute myocardial infarction or unstable angina had negative Tc-99m PYP scans. Although in our study Tc-99m PYP scanning was generally specific for acute myocardial infarction, this specificity has been questioned. In our study, 29% of patients who were diagnosed as having unstable angina without evidence of myocardial infarction, had a positive Tc-99m PYP scan (Fig. 1D). In addition, false-positive pyrophosphate scans have been reported in patients with stable angina,[6] ventricular aneurysms,[7] calcific valvular heart disease, mitral valve prolapse syndrome, acute pericarditis and following D.C. conversion.

The significance of a persistently positive Tc-99m PYP scan (one which can be demonstrated on repeat injection and scanning for more than six weeks) after myocardial infarction has also been reported.[8] Fifty-six percent of 109 patients were found to have a positive scan 6-288 weeks after a myocardial infarction. At the time of follow-up, 49.8 $\pm$ 21.1 weeks after the scan, the group with the persistently positive scan had a significantly increased incidence of severe angina, congestive heart failure, non-fatal acute myocardial infarction, and cardiac death. Further work is needed to confirm these findings.

At the present time, Tc-99m PYP scanning offers a means for the detection of acute myocardial infarction. It may be helpful in detecting myocardial infarction in situations where standard methods of infarct detection, i.e., ECG and cardiac enzymes, are nondiagnostic for an acute event. Thus, a patient with left bundle branch block or one who has a permanent pacemaker may sustain a myocardial infarction without a change in his electrocardiogram and a patient first seen 48 hours after a myocardial infarction may not have an elevated creatine phosphokinase (CPK) level. A positive Tc-99m PYP scan would aid in the diagnosis of myocardial infarction in these situations. The Tc-99m PYP scan has also been reported to be helpful in the detection of myocardial infarction following coronary artery by-pass surgery.[9] However, the apparent lack of specificity and the possible lack of sensitivity of this procedure may limit the usefulness of the pyrophosphate scan.

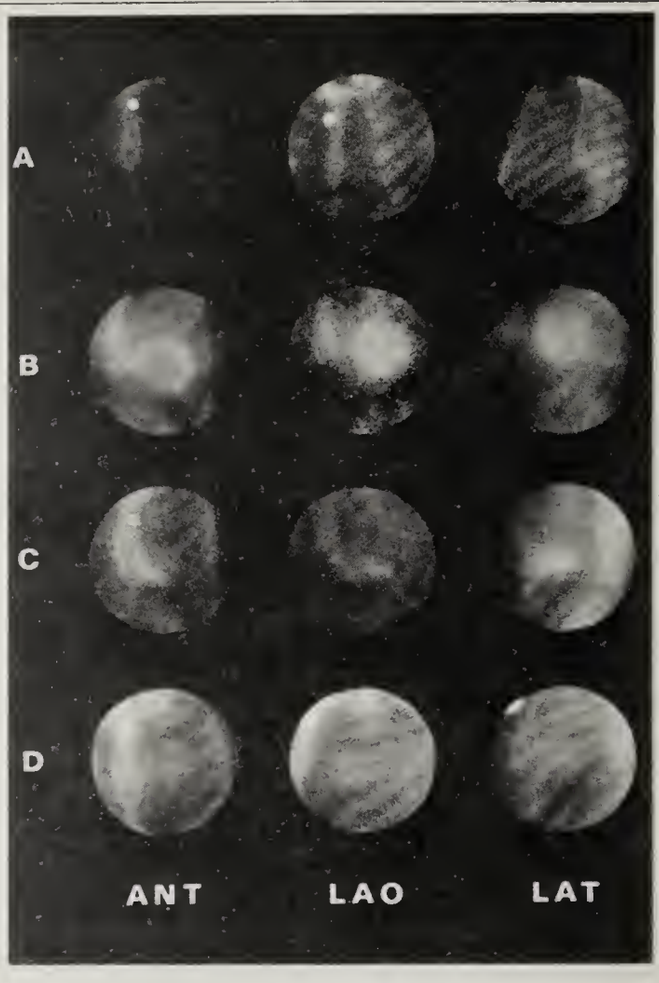


FIGURE 1

Technetium-99m pyrophosphate scintigrams in four patients with chest pain. (A) Technetium-99m pyrophosphate is taken up by the ribs and sternum without uptake of the radionuclide by the myocardium in a patient with chest pain but without evidence of myocardial infarction. (B) Technetium-99m pyrophosphate is localized to the anterior wall of the myocardium in a patient with an acute anterior myocardial infarction. (C) The radionuclide is localized to the inferior wall of the myocardium in a patient with an acute inferior myocardial infarction. (D) Localization of technetium-99m pyrophosphate in the myocardium in a patient with unstable angina. ANT = anterior, LAO = left anterior oblique, LAT = left lateral. (Reproduced from *Cardiology* 62:305-314, 1977, with permission of S. Karger, A.G. Basel).

MYOCARDIAL PERFUSION IMAGING

Thallium-201 is a potassium analog radionuclide which accumulates maximally in normally perfused myocardium after an intravenous injection. The uptake of thallium by the myocardium is a function of myocardial blood flow; thus, areas with preserved flow will appear as bright areas on the scan, while areas of hypoperfusion or scar tissue will appear as defects in the bright image (Figs 2 & 3). Thallium-201 is rapidly distributed in the myocardium and produces a stable scan for at least one hour after injection.[10]

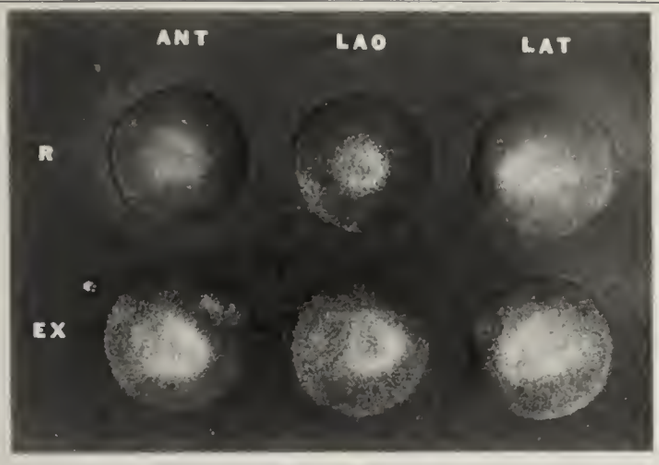


FIGURE 2

Thallium-201 myocardial perfusion scan showing no perfusion defect at rest (R) or with exercise (Ex); a negative thallium-201 stress test. ANT = anterior, LAO = left anterior oblique, LAT = left lateral.



FIGURE 3

Thallium-201 myocardial perfusion scan showing a defect developing during exercise on the anterior view. Arrows indicate the site of the defect; a positive thallium-201 stress test. Ex = exercise, R = rest.

Thallium-201 has been used primarily in the evaluation of ischemic heart disease. In a large series of patients with acute myocardial infarction,[11] 82% were found to have myocardial perfusion defects in their thallium-201 image. The incidence of defects was greater in those patients imaged within 24 hours of infarction, and all patients imaged within 6 hours had defects demonstrated. In those patients imaged serially, the size of the perfusion defect decreased with time. However, the major limitation to the use of thallium-201 in the diagnosis of acute myocardial infarction is its inability to distinguish acute ischemia or old infarction from acute myocardial infarction. One of the most promising features of thallium-201 perfusion imaging is its ability to detect areas of ischemic myocardium during exercise which are normal at rest. During an exercise test, thallium-201 is injected intravenously at the time when the patient experiences angina pectoris or severe fatigue. Exercise is continued for 30-45 seconds following the injection to allow for adequate distribution of the radionuclide. Imaging is performed 10-15 minutes after completion of the exercise test. If the exercise thallium scan shows uniform distribution of the radionuclide (Fig. 2), it usually indicates normal perfusion and there is little need to perform a resting study. If the exercise image shows a perfusion defect (Fig. 3), it must be compared to the resting image since thallium-201 cannot distinguish between acute ischemia and old myocardial infarction. This resting image can be obtained 4 hours after the exercise test without the need for reinjection of the radionuclide.[12] Perfusion defects detected after exercise, but not present at rest, are consistent with myocardial ischemia. Defects which are present at rest and do not change with exercise probably represent prior myocardial infarction and defects which are present at rest and increase during exercise are consistent with prior myocardial infarction and superimposed ischemia. A multicenter study for the evaluation of rest and exercise thallium imaging is presently underway. Preliminary data[13] suggest that the exercise thallium image combined with the exercise electrocardiogram has an increased sensitivity for the detection of significant coronary artery disease when compared to exercise electrocardiography alone. However, at present there is little data available concerning the ability of thallium-201 myocardial perfusion imaging to separate those patients with single, double, or triple vessel coronary artery disease or those patients with left main coronary artery disease.

Two major problems limit the usefulness of thallium-201 perfusion imaging. The first is the availability of the radionuclide. Few radiopharmaceutical firms are presently producing thallium-201 and the demand for this radionuclide has been great. The half-life of 74 hours for thallium-201 necessitates that new supplies of the radionuclide be obtained frequently and we have found that the agent is not always available when needed. The second major problem concerns the cost of the thallium scan. A rest and exercise thallium study costs the patient between $300-$400 in addition to the cost of the exercise electrocardiogram. For these reasons, we would recommend that the clinical use of thallium-201 perfusion scans be limited to the evaluation of those patients where the diagnosis of ischemic heart disease remains equivocal or where the etiology of a chest pain syndrome remains unresolved even after a treadmill exercise test has been performed. At our institution the thallium-201 perfusion scan has proved to be of particular value in those patients who have non-diagnostic exercise electrocardiograms.

EVALUATION OF CARDIAC PERFORMANCE

At the present time, two radionuclide techniques are available for the evaluation of left ventricular function. Both techniques require sophisticated data analysis using computer techniques. The first pass technique collects radioactive counts during a single passage of the radionuclide through the left ventricle for the determination of left ventricular ejection fraction. The gated cardiac blood pool technique allows collection of data from multiple cardiac cycles for the determination of left ventricular ejection fraction and for the evaluation of regional left ventricular wall motion.

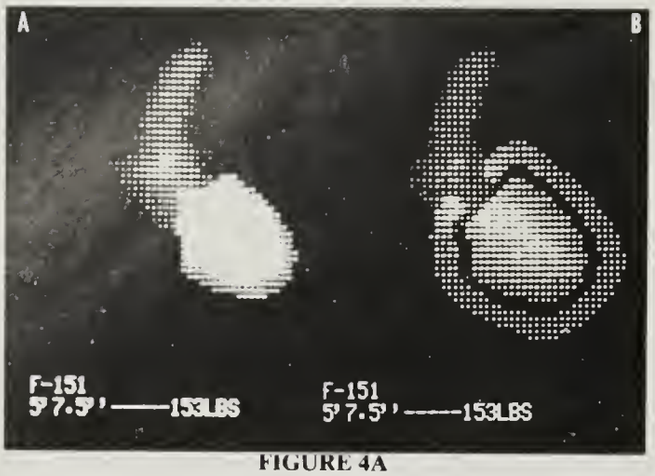


FIGURE 4A

Region of interest corresponding to the left ventricle is outlined using a light pen. See text for details.

FIGURE 4B

A background area is generated by encircling the left ventricle except for the area of the aorta.

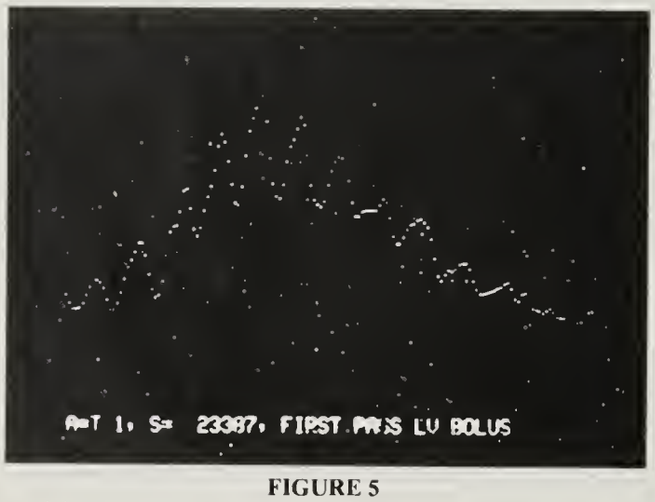


FIGURE 5

Time activity curve, corrected for background activity, from the left ventricular region of interest. See text for details.

First Pass Radionuclide Technique

Almost any radionuclide which can be detected by a gamma camera and which does not diffuse out of the vascular space into the pulmonary parenchyma can be used for this procedure. The most frequently used radionuclide is 99m-technetium either unbound or bound to albumin or DPTA. The radionuclide is injected into a large peripheral or central vein as a bolus followed by a saline flush. Radionuclide count rates are accumulated beginning with the injection of the radionuclide and are stored on a mini-computer disc. These data can be displayed on an oscilloscope and the passage of the radionuclide can be followed from the venous system into the right ventricle, through the lungs, and into the left ventricle and the aorta. A region of interest corresponding to the left ventricle is outlined using a light pen (Fig. 4A). A background area is generated by encircling the left ventricle, except for the area of the aorta, with the background being separated from the left ventricle by two matrix points (Fig. 4B). A time activity curve is generated

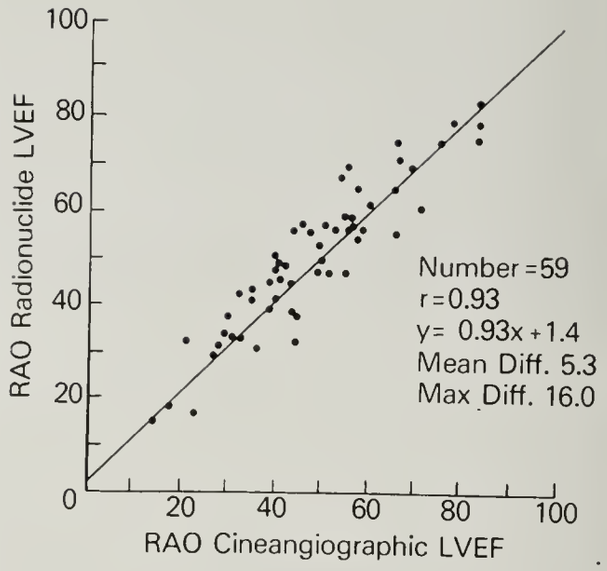


FIGURE 6

Correlation of left ventricular ejection fraction determined by the first pass radionuclide technique with the ejection fraction determined at the time of cardiac catheterization. LVEF = left ventricular ejection fraction. RAO = right anterior oblique.

for the left ventricular region of interest and for the background area. The left ventricular time activity curve, corrected for background activity, is obtained (Fig. 5). This curve is similar in appearance to a dye dilution curve, however, changes in count rate corresponding to systole and diastole of each cardiac cycle can be seen superimposed on the curve. The peak points of each cycle correspond to the activity at end diastole and the valley points correspond to the activity at peak systole. Radionuclide ejection fraction is determined by subtracting the counts at peak systole from the counts at end diastole and dividing by the counts at end diastole.

The first pass technique permits the accurate determination of left ventricular ejection fraction. Figure 6 shows the correlation of left ventricular ejection fraction determined by the first pass radionuclide technique with the ejection fraction determined at the time of cardiac catheterization in 59 patients from our institution. Excellent correlation is demonstrated over a wide range of ejection fractions. This high degree of correlation agrees with previously reported studies.[14]

The advantage of this method is that the data are obtained from a time activity curve and are not dependent on left ventricular geometry. Therefore, this technique is ideally suited for assessing function in the ventricle with segmental disease as seen in coronary artery disease, and has been used to perform serial measurements of left ventricular ejection fraction early and late after myocardial infarction.[15] The major limitation of the technique relates to the relatively low count rates which are collected from the single pass of the radionuclide through the heart. Using conventional gamma cameras for imaging, the high resolution needed

for analysis of regional wall motion may not be present. A recent report[16] suggests that improved computer analysis of the first pass radionuclide count rates will allow accurate determination of left ventricular regional wall motion abnormalities. Further work is needed to confirm these data.

Gated Blood Pool Technique

The gated blood pool technique for the evaluation of left ventricular function differs from the first pass technique in that count rates are collected from the left ventricle during multiple cardiac cycles and summed to produce a single composite image. This technique allows for the acquisition of high count rates which results in high resolution images needed for the evaluation of regional wall motion.

The radionuclide must remain within the vascular space for an extended period of time since several hundred cardiac cycles are needed to obtain adequate images. The radionuclide which is presently used for this technique is 99m-technetium labeled either to albumin or to human red blood cells. The R-wave of the electrocardiogram triggers a gate in the scintillation camera, turning it on during selected portions of the cardiac cycle, usually at end diastole and peak systole. Multiple points throughout the cardiac cycle can be obtained by interfacing the scintillation camera to a computer, and these latter images can be viewed in real time in a manner similar to contrast cineangiography.[17]

The gated blood pool technique can be used to obtain left ventricular ejection fraction by calculating a time activity curve of count rates within the left ventricle or by tracing the border of the left ventricle at end diastole and peak systole and applying standard cineangiographic formulas. An assessment of regional wall motion can be made by superimposing the peak systolic image and end diastolic image. Excellent correlation has been reported for the percentage shortening of the long axis and minor hemiaxis of the left ventricle between the gated cardiac blood pool technique and contrast angiography. Also, accurate detection of areas of left ventricular akinesia and dyskinesia has been reported using the gated blood pool image.[18]

Recently, the gated blood pool imaging technique has been used to evaluate left ventricular function during exercise. Abnormalities of left ventricular function, not present at rest, have been detected during exercise in patients with coronary artery disease[19] and in patients with aortic insufficiency.[20] Further work is needed to evaluate the significance of these findings.

CONCLUSION

Cardiovascular nuclear medicine techniques are useful in the clinical evaluation of myocardial infarction, myocardial perfusion at rest and exercise, and myocardial function at rest and during exercise. Cardiovascular nuclear medicine techniques offer the practicing physician a wide variety of useful techniques for the evaluation of cardiovascular disease.

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Emotional Problems Of Physicians: II

Current Approaches To The Problem

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ABSTRACT

Two approaches for dealing with the emotionally ill physician have been offered: coercive and non-coercive. "Sick doctor statutes" provide measures to limit or halt practices while the physician is judged incompetent by virtue of his illness. Non-coercive measures include Physicians' Committees in medical societies consisting of experts in drug adiction, alcoholism, or mental illness who serve to help sick physicians get appropriate treatment. The issue of physicians incompetent to practice by virtue of an emotional illness is an ethical one for which the entire medical profession has a collective responsibility.

Whether the sick physician's problem is due to emotional and/or physicial causes, his illness creates a problem in medical ethics. The ethical problem is whether his illness interferes with his competence to practice medicine. Since the medical profession is collectively responsible for monitoring the practice of medicine, it is responsible for identifying the sick physician, limiting or temporarily halting his practice while he is ill, and recommending treatment. The ultimate responsibility for seeking and accepting treatment is the sick individual's, in this case the sick physician's.

Since the ethical problem of the sick physician is a collective one, steps for dealing with it have been proposed at local levels, principally through hospitals and local medical societies; at state levels through state medical societies often in conjunction with state medical examining boards, which have been legislated authority to issue licenses to practice and deal with ethical problems; and national levels, through the American Medical Association, particularly through its Council on Mental Health. The latter group has been charged with recommending a model law, the "sick doctor statute", for individual states to consider. The statutes adopted have coercive passages to limit or halt practice while the sick physician is judged incompetent.

The non-coercive measures are attempts to deal with the sick physician by helping him recognize that he has a problem and to guide him into voluntary treatment. They more often involve local groups, such as Physicians' Committees in local medical societies consisting of experts in drug addiction, alcoholism, or mental illness, who have no disciplinary or reporting powers. The State of Oregon has a group called the Friends of Medicine consisting of lay members as well as physicians, organized outside the structure of organized medicine, set up to help the sick physician.

The Sick Physician Statute

In 1969, the State of Florida passed the first sick doctor statute,[1] followed by Texas in 1971. An excellent review of this model law was presented by the Council on Mental Health of the American Medical Association.[2] The law establishes that the physician suspected of "inability to practice medicine with reasonable skill and safety to his patients due to organic illness, mental or emotional disorders, deterioration through the aging process, or loss of motor skill" comes under review. The inabilities to practice "can arise from excessive use or abuse of narcotics, drugs and chemicals, alcohol or similar types of material".

If probable cause is established then the involved physician is required to undergo diagnostic mental and physicial evaluations.

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Implied consent for such examinations is given when the physician renews his annual medical license.

Following a hearing, the Board of Medical Examiners has the power to suspend or limit practice, in addition to placing him on probation during the time he is ill and not capable of practice. Also, he may be compelled to seek therapy in order to retain or regain his license.

Such actions of the Board cannot be used against the physician in other legal proceedings.

The state medical boards are often assisted by committees of experts formed by the state medical society. In Arizona such a committee called "The Committee for Physician Rehabilitation" serves under the combined authority of the State Medical Board and State Medical Association.[3] It accepts referrals from the troubled physician himself, his family, colleagues, his hospital, his county medical society, or the State Board of Medical Examiners.

Though its prime orientation as stated is toward counsel and rehabilitation it may implement the authority of its two sponsors by "setting or recommending conditions, to be met by the doctor patient, limitations to his practice, or activities". This authority occurs in a state where reporting all physicians suspected of emotional problems to the State Board of Medical Examiners by another physician is mandatory under the law.

This mandatory reporting could make it difficult for the physician to seek voluntary, anonymous therapy in his own state. The process of recognizing one's own infirmities, knowing where to turn, and receiving appropriate therapy are difficult enough steps. The mandatory reporting may be, in some cases, self defeating by delaying the sick physician's seeking early help. Such delay may ultimately lead to increasing disability which then would require mandatory measures.

The Non-Coercive Approach

In San Francisco an Advisory Committee for Physicians was established to help physicians with emotional problems. Physician colleagues can report a physician in need help and, in turn, the committee members will contact the physician in question, confidentially, to try and help him.

The Medical Society in New York State has described two separate approaches to the problem.[4] The coercive approach is through legal channels via the Division of Professional Conduct of the Department of Education. This has yet to be implemented.

The non-coercive approach establishes a Physicians' Committee of the state medical society consisting of experts in alcohol, drug abuse, and mental illness. They serve without pay, remain anonymous, and do not serve on any disciplinary committees. A physician colleague can call the committee and identify a sick physician. If the caller wishes to remain anonymous then a second physician colleague must also declare the physician has a suspected illness. This is done to minimize harassment by a colleague. With two anonymous complaints, or one identified complainant, then the committee is activated. Two experts who do not know the person contact the alleged sick physician. Their sole purpose is to offer help if he wants it. If he wishes help, they will refer him to other experts for care. If he refuses, he is assured that he can contact the experts from the committee in the future should he change his mind. No written records are kept. The original complainants' names are kept confidential.

If the sick physician continues to refuse help and the colleagues originally concerned observe no change in his behavior and they deem prompt action necessary, they are free to initiate coercive actions through the established legal channels.

DISCUSSION

The final authority for the right to practice medicine resides in State Boards of Medical Examiners. Since ethical problems related to the physician's incompetence to practice due to drug addiction, alcoholism, and emotional illness come under scrutiny of these boards, model legislation has been offered by the American Medical Association to deal with emotionally sick physicians. These efforts are necessitated by the individual physician's denial of emotional problems and his failure to seek individual help.

Alternate non-coercive approaches by local medical societies, state societies, and state organizations are being tried to aid the physician in recognizing his disorder and facilitating his getting into treatment. These approaches are aimed at helping sick colleagues obtain voluntary care without the threat of disciplinary action. The mandatory reporting of all physicians suspected of emotional, drug, or alcohol related problems denies the possibility of a non-coercive approach.

Since all these approaches are new, it is difficult to assess their effectiveness. Physicians in all states are encouraged to support any approaches which will identify physicians in distress, work toward minimizing their suffering, while serving our collective professional responsibility to the public by restricting the practice of incompetent colleagues during the time they are impaired by virtue of their mental, or emotional disturbances. Ultimately, the treatment of the sick physician will spare him the embarrassment of public exposure of his symptoms, the shame of his family related to his inappropriate behaviors, and the guilt of his colleagues for not responding in his hours of grief.

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Lupus Glomerulonephritis

Short Review And Guide To Therapy

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Systemic lupus erythematosus (S.L.E.) is a multisystem disease of unknown etiology. It is often a benign condition, whose only manifestation may be intermittent arthralgias responsive to anti-inflammatory therapy. However, when the central nervous system or the kidneys are involved, the prognosis is less favorable and more aggressive therapy is required. In the past these were the leading causes of death in S.L.E., but with the widespread use of immunosuppressive therapy, infection is now the major mortality factor. Lupus nephritis is unique, in that untreated, it may progress to end stage renal failure requiring dialysis or transplantation, whereas with early diagnosis and aggressive therapeutic intervention, disease activity is controllable and the pathologic changes are reversible.

In patients with active S.L.E. the incidence of lupus nephritis is 50-60% based on clinical presentation and urinalysis. Signs and symptoms may be those of any acute glomerulonephritis and are not unique to S.L.E. They include elevated blood pressure, fluid retention leading to edema formation or pulmonary congestion, decreased urine output and the nonspecific symptoms associated with azotemia such as anorexia, nausea, vomiting, malaise, headaches, lethargy, etc. Urinalysis may reveal the presence of red blood cells, red blood cell casts, and proteinuria, suggesting glomerular inflammation. The so-called telescoped urine may contain, besides the cellular elements and cellular casts (both red blood cell and white blood cell casts) oval fat bodies, hyaline and broad casts as well as debris. However, urinalysis alone underestimates the incidence of renal involvement. Recent reports of kidney biopsies in patients with active S.L.E. but no clinical evidence of renal disease, have shown immune complex deposition by electron microscopy and immunofluorescence in 100% of cases.[1,2]

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TABLE 1

Histopathologic classes of lupus nephritis:

- Focal Mesangial Glomerulitis
- Focal Proliferative Glomerulonephritis
- Diffuse Proliferative Glomerulonephritis
- Membranous Nephropathy
- Interstitial Nephropathy

There is general agreement that lupus nephritis may be divided into at least four major categories based on histopathological findings (see Table 1).

1. **Focal mesangial glomerulitis:**

This may appear normal by light microscopy or have mild focal increase in mesangial cells or matrix. There is no inflammatory response, leukocytic infiltration or necrosis. By immunofluorescence and electron microscopy, deposition of immunoglobulin and complement occurs only in the mesangium. These individuals may have normal glomerular filtration rates. Mesangial lupus glomerulitis is considered to represent the earliest stage in the development of lupus nephritis.

2. **Focal proliferative glomerulonephritis**

This lesion accounts for approximately 30% of all cases of lupus nephritis. The lesion is focal, with some glomeruli being completely normal and others showing involvement of only some lobules, i.e., segmental distribution. There is proliferation of mesangial and endothelial cells with variable exudation, neutrophilic infiltration, and necrosis. Glomerular sclerosis occurs in a small percentage of glomeruli and may be segmental or global. Immunofluorescence reveals immunoglobulin, complement and fibrin predominantly in the mesangial zones, but occasionally involving all the peripheral capillary loops. Even in areas which on light microscopy appear normal, immune complexes can be demonstrated with electron microscopy and immunofluorescence.

3. **Diffuse proliferative glomerulonephritis**

This is the most common form of renal involvement in lupus accounting for greater than 50% of cases. The process is diffuse involving all the glomeruli with proliferation of endothelial and mesangial cells as the predominant feature, in association with exudation, neutrophilic infiltration and tuft necrosis. In severe cases epithelial cell proliferation appearing as crescent formation gives rise to a rapidly progressive glomerulonephritis. Immunofluorescence reveals immune complex deposition throughout

the mesangial zones and along the peripheral capillary loops in a subendothelial location. The extensive subendothelial deposition of immune complexes and fibrin gives rise to the "wire loop" lesion seen by light microscopy. Electron microscopy confirms extensive immune complex deposition, inflammatory injury, and necrosis.

4. Membranous glomerulonephritis

This is the least common manifestation of lupus nephritis and accounts for 10-15% of the total cases. The light microscopic features are identical with those of idiopathic membranous glomerulopathy. Usually there is no cellular proliferation or evidence of inflammatory injury in the glomerulus. Immunofluorescence reveals discrete granular deposits containing immunoglobulin and complement along the basement membrane. The occurrence of IgA in these deposits is highly suggestive of lupus, being very uncommon in idiopathic membranous nephropathy. Electron microscopy reveals intramembranous and epimembranous deposits, variable glomerular basement membrane thickening and new membrane formation.

In association with these subtypes of lupus glomerulonephritis, there is variable involvement of the vasculature and the tubular-interstitial structures. The small arterioles may show myointimal thickening, intramural hemorrhage and occasionally necrotizing vasculitis with luminal occlusion. Granular deposition of immunoglobulin and complement is an occasional finding in the vessel wall. Renal interstitial cellular infiltrate and tubular necrosis may occur in a focal patchy distribution and tends to parallel the degree of glomerular involvement. Occasionally interstitial disease is the predominant feature and may be associated with immune complex deposits in the interstitium and along the tubular basement membrane.[3]

The clinical features of lupus nephritis correlate in part with the pathologic changes. In the mildest form, focal mesangial glomerulitis, microscopic hematuria, low grade proteinuria (less than 1 gram per day), in association with normal renal function is usual. In focal proliferative nephritis increasing proteinuria is found (1-2 grams per day), patients are rarely nephrotic, and a modest decrease in renal function may occur. In diffuse proliferative disease most patients have nephrotic range proteinuria (greater than 3.5 grams per day) and diminished glomerular filtration rate. In severe cases deterioration of renal function may be rapid and progressive, especially when vasculitis, necrosis and crescent formation are predominant features. Diffuse proliferative nephritis is a progressive disease which, if left untreated, tends to result in renal failure in less than three years.[4] Membranous nephropathy presents initially with nephrotic syndrome and occasinally with mild azotemia. Although remission occurs in about one-third of patients, this disease tends to be a slowly progressive disorder terminating in renal failure.[4]

Patients with systemic lupus who have no evidence of renal disease at the time of initial presentation tend not to develop nephritis at a later time, although there are well documented exceptions to this rule. In the patients with established renal disease, it used to be thought that the initial biopsy was a good prognostic indicator, in that the individual patient would not change from one histopathologic subclass to another. However, this is now known to be untrue. Patients with mild disease (i.e., focal proliferative or mesangial nephritis) may progress if untreated to diffuse proliferative nephritis.[5,6] Thus, initial biopsy classification although very helpful in determining a therapeutic strategy does not alone provide a definitive prognosis.

The etiology of systemic lupus is not understood but much is known about the events leading to end-organ damage. The normal individual has an inherent mechanism which prevents antibody production against normal body constituents. In S.L.E. this normal regulatory ability fails and antibodies are produced against a variety of intracellular and membrane antigens. The hallmark of the disease is the development of autoantibodies against several nuclear constituents, including native DNA, RNA and nuclear proteins. The production of autoantibodies leads to formation of circulating immune complexes, which are subsequently trapped in the various capillary beds of the kidney, skin, central nervous system, joints, etc. The physical characteristics of these immune complexes, i.e., their size, conformation, antigen-antibody ratio, electric charge, are very important in determining their localization, their uptake by the cells of the reticuloendothelial system, and their ability to incite inflammatory response, or activate the complement system.

Lupus glomerulonephritis can be viewed as a continuum with mesangial involvement as its earliest manifestation. With increasing quantities of autoantibody formation, the ability of the kidney to dispose of these deposited immune complexes may be overwhelmed. Subsequently, with complement activation and neutrophilic response, the glomerular inflammatory injury becomes widespread. Thus, focal disease would tend to become diffuse proliferative, if immune complex deposition is unchecked. Membranous nephropathy, with absence of inflammation and proliferation, is a distinct entity, with evidence for a difference class of antibody deposition.

In practical terms, the fluorescent antinuclear test (FANA) provides a useful indicator of autoantibody production. However, positive ANA can occur in many conditions including other collagen vascular diseases, liver diseases, and following certain drugs. Nevertheless, antibody to native DNA giving a rim pattern on the FANA is fairly specific for S.L.E. There is a positive correlation between clinical activity of S.L.E., anti-DNA antibody titers, and complement activation.[7] Careful monitoring of these parameters is helpful in treating the active phase of the disease or predicting clinical exacerbations.

TREATMENT

Diffuse proliferative nephritis, if untreated results in irreversible renal failure; therefore, early diagnosis and therapy are of the utmost importance.[4] The aims of therapy are twofold:

(1) Decrease the inflammation, proliferation and exudation in the kidney; and (2) Induce a serologic remission of the systemic disease — i.e., reduced autoantibody production and complement activation.

Evaluation of a patient with lupus and suspected nephritis should include a detailed history and physical examination (see Table 2). A family history, history of drug ingestion, sunlight exposure, and recent infections should all be included. Following clinical assessment of S.L.E. activity (arthalgia, rash, alopecia, serositis, CNS complaints), a serologic evaluation including ANA, serum complement, Coombs test, VDRL, rheumatoid factor and cryoglobulins should be obtained. Hematologic evaluation includes a CBC, platelet count, and coagulation profile. Renal function is assessed by BUN, creatinine, and 24 hour urine for creatinine clearance and total protein excretion. The urine sediment is analyzed for cells, casts, bacteria, etc. An intravenous pyelogram may be obtained for evaluation of renal size and anatomy. Finally, a kidney biopsy is performed, and the tissue studied by light, immunofluorescence and electron microscopy. The importance of the kidney biopsy is in providing an important baseline parameter which enables the physician to direct initial therapy, monitor its effectiveness, and aids in overall patient management.

Corticosteroids are the mainstay of therapy in lupus nephritis. They possess both anti-inflammatory, antiproliferative and immunosuppressive properties. Early studies indicated that low dose Prednisone was ineffective in treating proliferative nehritis whereas higher doses (60-100 mg. per day) definitely altered the clinical outcome of the disease.[8] Unfortunately many of the reports of therapy in lupus nephritis deal with small numbers of patients, are uncontrolled, and make no attempt to differentiate the various pathologic subtypes. However, most workers agree that in patients with focal proliferative nephritis, Prednisone 60 mg. a day (or 1 mg/Kg) is a reasonable initiating dose. This will usually also control the extrarenal manifestations of the disease. Monitoring renal function, proteinuria, hematuria, as well as ANA titers and serum complement levels should be undertaken during therapy and follow-up. Generally after two to three weeks, if it can be shown that serology and renal function are approaching normal, the dose may be decreased and gradually changed to an alternative day regimen. When the disease becomes serologically inactive, low dose alternate-day therapy may be sufficient to maintain a remission.

High dose Prednisone has been used successfully to treat diffuse proliferative lupus nephritis, but the significant side effects of long term high dose Prednisone has prompted an evaluation of cytotoxic drugs as alternative or adjunctive therapy. Cyclophosphamide, Azathioprine, and Chlorambucil have all been used in this disease. There is considerable controversy as to their value. Reports are often anecdotal and even when controlled studies have been done, results have been contradictory.

TABLE 2

Evaluation of the patient with lupus nephritis

I. History
II. Physical Examination
III. Hematology: CBC, platelet count, coagulation profile
IV. Serology: ANA, C_3, C_4, Coombs Test, VDRL, Cryoglobulin, Rheumatoid Factor
V. Renal: BUN, creatinine, 24 hr. urine for creatinine clearance and protein excretion, urinalysis
VI. IVP, Renal Biopsy (light, electron microscopy and Immunofluorescent studies)

Steinberg[9] and Cameron,[10] in short term studies of less than one year, suggested that Cyclophosphamide was a useful adjunct to Prednisone, and Donadio[11] reported a decrease in disease progression over a two year follow-up period. However, the toxic side effects of this drug including leukopenia, alopecia, and hemorrhagic cystitis have precluded long term therapy, which is required in lupus.

Azathioprine has received the most extensive use of all the immunosuppressant agents. Donadio,[12] Hahn[13] found no overall advantage when Azathioprine was added to Prednisone over a three year follow-up. Decker[14] using high dose Azathioprine had a high incidence of toxic side effects, and additionally found no improvement over Prednisone alone in the outcome of lupus nephritis. Small numbers of patients, variable dosage regimen of Prednisone or Azathioprine, ethnic differences in patient populations, and lack of statistical significance must be considered in evaluating and comparing these reports. In contrast, Cade,[15] using Azathioprine with heparin or Prednisone, and Sztejnbok,[16] using Prednisone and Azathioprine, both encountered decreased patient mortality from lupus nephritis during a four year follow-up. Recent studies with large groups of patients have clearly shown that Prednisone-Azathioprine combination is effective in decreasing morbidity and mortality in long term follow-up (up to 17 years).[17,18] Patients with milder renal involvement progress less frequently to more severe forms when treated with Azathioprine and Prednisone, when assessed by sequential renal biopsy. It is also apparent that with combined drug therapy, lupus patients tend to have a more benign clinical course, fewer exacerbations and less overall hospitalization time.

Thus, with diffuse proliferative disease, the use of Prednisone in combination with Azathioprine seems appropriate. Its immunosuppressant properties, steroid sparing qualities and minimal side effects make Azathioprine an important adjunctive agent. Administration of Azathioprine to patients should only be undertaken with a detailed knowledge of its potential toxic effects which include bone marrow depression, leukopenia and liver dysfunction. Infection, often with an opportunistic or resistant organism, is now the major cause of death in

lupus, and is directly related to widespread use of these immunosuppressive agents.

A recent report has suggested the use of "pulse therapy" — Solumedrol/1 gram/day for three successive days in the treatment of active lupus nephritis. In patients who are steroid toxic, and cannot tolerate long-term daily steroids, this may provide a useful alternative. It is not recommended as standard therapy at this time.[19]

Plasmapheresis has been utilized in the therapy of rapidly progressive nephritis and Goodpasture's disease, a condition where removal of antibody or immune complexes is beneficial. In a small series of lupus patients with high levels of circulating immune complexes, plasmapheresis was responsible for a significant clinical and serologic improvement. This relatively benign procedure may hold promise for future therapy.[20]

Membranous nephropathy in S.L.E. is a variable disease. There are no controlled studies to determine optimal therapy, but two drugs are generally recommended. The response to drug therapy is unpredictable and the nephrotic state occasionally remits. Progression of the renal disease may be dependent on systemic serologic activity. Renal failure which is often slowly progressive is generally a reflection of glomerulosclerosis, and is obviously irreversible.

Continued surveillance of patients with S.L.E. nephritis is an important part of their care. This is a lifelong disease which is controlled but not cured by medication. Routine clinical follow-up and regular serologic evaluation including complement levels and ANA titers are important. A rise in ANA titers, or a change in ANA patterns or a decreasing serum complement will frequently precede and predict a clinical relapse. However, it is not suggested that an abnormal laboratory value be the sole criterion for treatment. It should serve to alert the physician that the disease is active and immune complex formation is occurring, and enable preventive action to be undertaken.

When decreasing or discontinuing immunosuppressive medications, the possibility of relapse should be considered. It is apparent that when Azathioprine is stopped in a clinically stable patient, relapse may occur, often resulting in irreversible renal deterioration or reduced sensitivity of the renal lesion to immunosuppressant therapy.[21]

SUMMARY

It has been seen that lupus glomerulonephritis is not a single entity. There are several histopathologic subtypes, which may be viewed as a continuum, with focal proliferative nephritis as the earliest lesion and diffuse proliferative nephritis as the most advanced. Membranous glomerulopathy appears to be a distinct subtype, with evidence for a different class of autoantibody formation. Clinical presentation is of limited value in determining the renal lesion, and kidney biopsy is critical in planning therapy. Antinuclear antibody testing and serum complement levels are important in assessing the serological activity of lupus, and a changing serology will often predict a clinical relapse. Aggressive immunosuppressive therapy can reverse the histopathologic lesions of lupus nephritis, and prevent recurrence. Careful monitoring of clinical symptoms, serology and kidney function allow for an excellent prognosis. With the adjunct of renal replacement modalities such as dialysis and transplantation, death from renal failure in S.L.E. should be almost completely avoidable.

ACKNOWLEDGMENTS

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Herpes Simplex:

Epidemic Unchecked

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In direct relation to patients' mounting expectations of the medical community to provide cures for diseases of all forms, the clinician's frustration in dealing with any condition that is becoming more common, while remaining essentially untreatable, has also intensified. Herpes simplex represents such an entity. Partially as a result of changes in social customs in recent years allowing for increased physical contact, the incidence of this painful and socially-disabling affliction has reached near-epidemic proportions. Unfortunately, advances in therapy have not paralleled the explosion in occurrence, and treatments continue to be either symptomatic or based on anecdotal reports, unconfirmed by consistent clinical data.

Herpes simplex is actually a spectrum of infections caused by *Herpesvirus hominis*, a spherical or icosahedral DNA virus approximately 180 mm in diameter.[1] *In vivo* the virus replicates within the nucleus of infected cells, and the growth cycle may be as short as five hours. Using sophisticated cultural and laboratory criteria, the virus can be classified into two types: Type 1 (HSV-1) which clinically is seen infecting areas above the waist such as the mouth, face, eye and nose and is usually responsible for cases of eczema herpeticum; and Type 2 (HSV-2) which characteristically produces a vesicular eruption of the genitalia, anus, sacrum and buttocks and has been incriminated as a possible contributing factor to cervical cancer.[2,3]

EPIDEMIOLOGY

The magnitude of the present herpes simplex epidemic is brought into focus when one considers the fact that an estimated one-third of the world's population is now infected with HSV-1, and a much smaller fraction infected with HSV-2.[4] Because the virus has the ability to persist lifelong within the infected person, a reservoir of virus for dissemination is permanently maintained. Unfortunately, acquired immunity to one type of HSV does not protect against infection with the other. This fact combined with the increasing practice of oral-genital contact has set the stage for concurrent infections with both types. Further compounding the problem, asymptomatic carriers, found to be as high as 15% of males in one series,[5] periodically shed infectious viral particles infecting unsuspecting new contacts. Presently, only gonorrhea exceeds HSV as a cause of venerally-spread disease.

Corresponding to the two types of HSV, two epidemological patterns were initially recognized with HSV-1 commonly appearing above the waist and HSV-2 below. However, these distinct patterns are now becoming blurred as an increasing number of genital lesions, 50% in one clinic,[6] are now caused by HSV-1 acquired by oral-genital contact. Likewise, some "cold sores" of the lip are now caused by HSV-2 acquired in the same way. Consequently, a clinician can no longer determine with certainty which type of HSV might be responsible for a particular lesion, based on body location alone. On the other hand, some generalizations are still valid. Most herpetic lesions about the mouth are acquired non-venerally, and most genital herpes is acquired by sexual contact. Occasional genital lesions, especially those seen in children, may be HSV-1, acquired by autoinoculation.

HSV is acquired by direct contact with the active virally-infected lesions of another. Intact skin poses a barrier to infection, but broken skin and mucous membranes offer little resistance to infection. Once gaining access, the virus incubates an average of 6 days, at which time a lesion appears at the site of entry. This primary infection may take one of various forms. In the case of HSV-1, which is commonly acquired before the age of 5, the primary infection may be subclinical or appear as a few small transient ulcerations in the mouth, rarely as a severe gingostomatitis.

Similarly, the primary infection with HSV-2, can vary from subclinical, to a few small genital ulcers in men, to a severe vulvo-vaginitis in women. In exceptional cases, the HSV may gain access to the central nervous system, either by way of peripheral nerves or blood vessels, during the primary infection and result in a potentially fatal encephalitis or meningitis.

Following the primary infection, which heals in 8-14 days, and is usually more intense than subsequent recurrences, antibody formation begins, and a period of latency commences. Unfortunately, little cross protection is afforded by these antibodies, and even high titers of HSV-1 antibodies do not preclude a primary infection with HSV-2, although such an infection may be clinically milder than in a totally unprotected host.[7] The location of HSV between recurrent attacks is still a matter of controversy. All attempts to recover it from sites of recurrence have been unsuccessful, but recent studies[8,9] have demonstrated the virus in the spinal root ganglion during periods of latency. It is thought that during the primary infection, the HSV ascends the sensory nerve fibers to the level of the spinal root ganglion. Here it remains in a non-replicating, non-infective form, until a

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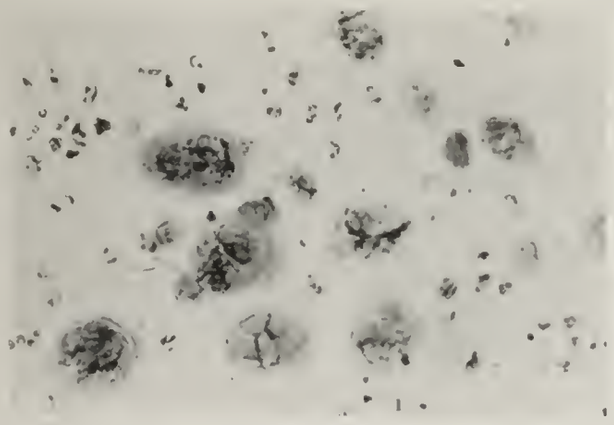


FIGURE 1
Tzanck smear showing multi-nucleated giant cells (modified Papanicolaou stain, original magnification X450).

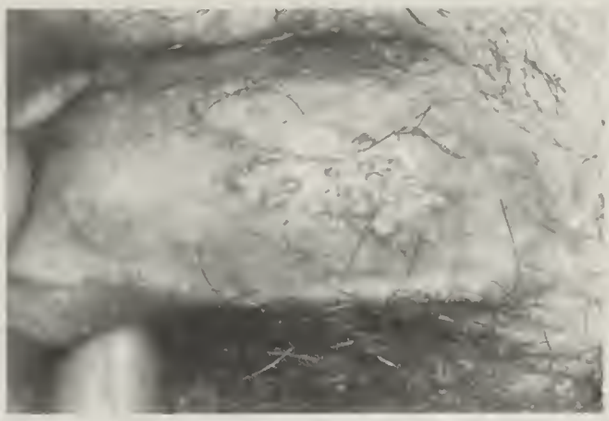


FIGURE 2
Clustered vesicles of herpes simplex on penile shaft.

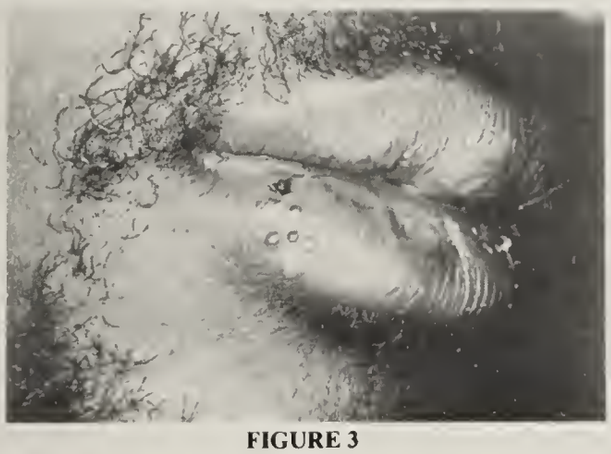


FIGURE 3
Herpes labialis.

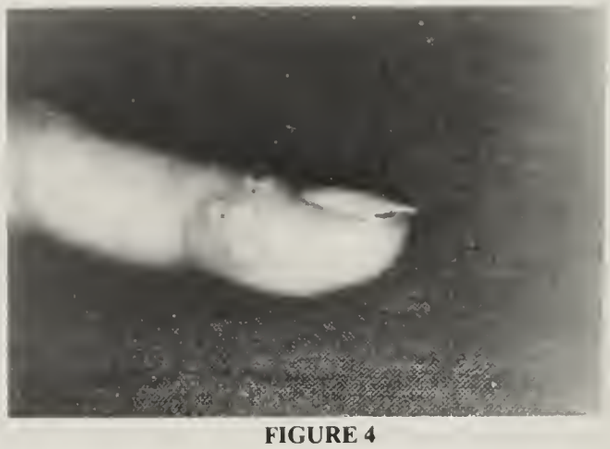


FIGURE 4
Herpetic whitlow.

disturbance in the status quo at the ganglion causes the release of a portion of the virus to be shed back down the nerve to the approximate site of the initial lesion. The body's natural defenses responsible for terminating initial and subsequent attacks and maintaining symptom-free periods between recurrences is not fully known, although cell-mediated immunity presently appears to play the most important role in this defense.

CLINICAL PRESENTATION AND DIAGNOSIS

As previously mentioned, the primary HSV infections may vary from subclinical to a devastating gingostomatitis or vulvo-vaginitis accompanied by fever, regional lymphadenopathy, and other constitutional symptoms. When presenting in this latter severe form, the clinical impression, while suggestive, cannot be definitely confirmed without viral culture. Tzanck smears taken from the base of an active lesion may be helpful in confirming the diagnosis by demonstrating multi-nucleated giant cells (fig. 1) which occasionally contain identifiable viral inclusion bodies. This simple test using readily available stains such as Wright's or Giemsa also has the advantage of providing diagnostic information immediately.

The recognition of recurrent lesions of HSV infection is relatively easy. After a transient prodrome of numbness, burning, tingling, or pain, approximately 6-10 small, clear vesicles appear on an erythematous, slightly edematous, base. (Fig. 2, Fig. 3) The vesicles rapidly become pus-filled and cloudy, easily breaking with any manipulation. Intact vesicles or pustules in herpes progentalis are less commonly observed than the resultant grouped, shallow, erosions. Occasionally, these individual lesions may coalesce forming a shallow ulcer which must be distinguished from other conditions, particularly the syphilitic chancre. Another presentation is called the herpetic whitlow (Fig. 4) in which tender, flat, grouped vesicle-pustules are seen on the finger. This is a form of HSV acquired by members of the medical, dental, and nursing professions, by contact with patients' active lesions. Once acquired, these painful recurrent lesions serve as a ready source of infection to others.

PREVENTION AND TREATMENT

Considering the difficulty to date in finding an adequate treatment for herpes simplex, prevention of dissemination to new hosts assumes particular importance. Recommendations include general advice appropriate for various microbial infections and specific precautions required for the unique properties of HSV. Since prevention of primary infection of necessity often calls for some period of abstention from direct physical contact, patient acceptance is occasionally lacking. Fortunately, recent clinical studies have shown that the virus is shed from active lesions for only a maximum of four days,[10] corresponding to the progression of the lesion from a vesicle to a crust or shallow ulcer. Consequently, during the infective vesicular stage, all direct contact should be avoided. Depending on the locations of involvement, this

"contact quaranteen" may constrain the patient from kissing for four to six days or may allow intercourse only with the protection of a condom. In addition, immunesuppressed individuals, newborns, and anyone with atopic dermatitis has increased susceptibility and should be protected from exposure to HSV.

Prevention of recurrences is only as successful as the ability of the patient to control the circumstances which trigger subsequent episodes. Prolonged exposure to sunlight should be avoided, with a sunscreen containing para-aminobenzoic acid (PABA) applied to uncovered skin especially that of the lips before any extended periods of solar exposure. Trauma, emotional stress, and fever may be more difficult to avoid. Patients may be unable or unwilling to avoid other causes of recurrences such as menses and intercourse.

Despite intermittent reports of new forms of therapy, the availability of any unequivocally effective approach to treatment is lacking. To be effective, an agent must be able to preferentially destroy a DNA-containing virus which is replicating in the nucleus of a host cell without damaging or altering the natural DNA, and must be able to reach all infected cells. Such an agent has, as yet, not been found. Moreover, the natural course of the clinical disease complicates controlled studies because of its variable duration and severity, independent of treatment.

Some of the older modes of therapy are now contraindicated because of risk to the patient. Small pox vaccinations, for example, while anecdotally helpful in increasing the interval between recurrences, expose the patient, his family and friends to the possibility of generalized vaccinia. Similarly, the use of various photoactive dyes followed by exposure to visible light has not been proven effective in well-structured studies[11] and may conceivably be carcinogenic in some patients.[12] This latter assertion, however, is a matter of controversy since lesions of squamous cell carcinoma of the penis have now also been demonstrated in young men who have not had this form of therapy.[13]

Desicating agents may be useful in bringing about a quicker resolution to the symptomatic vesicular stage, although not actually irradicating the virus from the lesion. Ether may work well in this way, but is extremely volatile and highly flammable. Consequently, its use in office or home is attended with an element of real danger. Recent investigations at Baylor University have shown both a decrease in viral activity and in lesion healing time using either 4% thymol in chloroform or 2% buffered gluteraldehyde (Jarratt, M: personal communication). However, the former of these solutions has an unpleasant odor and may cause a superimposed irritant dermatitis, while the latter stains the skin brown and remains stable for less than one week.

Certain agents currently hold some promise in that they may have selective activity against the virus. Phosphonoacetic acid has been shown to shorten clinical infections in mice if applied shortly after exposure to HSV.[14] This chemical binds to active sites of DNA polymerases produced by the virus rather than to enzymes of the host cell, but as yet is unwieldy to use.[15] Butylated hydroxytoluene (BHT) is safe for mammalian cells and will inactivate certain lipid-containing viruses including HSV *in vitro*.[16] BHT has been used in a 5% concentration clinically but is not readily available in this country through normal pharmaceutical channels. The antiviral drugs, idoxuridine and vidaribine, have been proven effective against HSV in the eye.[17] However, neither has as yet been demonstrated to be of value in cutaneous infections, nor approved for such use by the Food and Drug Administration. The package insert for vidaribine (Vira-A Ophthalmic Ointment, 3% - Parke-Davis) describes evidense of teratogenicity in laboratory animals, and warns against use in pregnancy. The mutagenic capability of these medications when applied to the mucous membranes of the genital area must also be taken into consideration.

The brightest hope for the future containment of this present epidemic would seem to lie in the field of immunology which has enabled us to conquer so many of the viral scourges of the past. At present, the three commercially available vaccines prepared from inactivated HSV are not consistently effective in preventing either primary or recurrent infections. In addition, there is a risk that even inactivated virus may be capable of stimulating dysplasia through nucleic acid transformation in mammalian cells.[2] Hopefully, a vaccine will some day be perfected with immunogens which contain no viral-DNA but are capable of specifically inducing immunologic memory, permitting a protective host response on subsequent exposure to the virus. Such a development would be gratefully welcomed by disillusioned patients and frustrated practitioners alike.

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Advances In The Classification Of Acute Leukemia In Children

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Frank L. Meyskens, M.D.

Recent therapeutic advances have led to an improved outlook for children with acute lymphoblastic leukemia (ALL), the most common form of leukemia in children (Table 1). The development of effective combination chemotherapy regimens for both induction and maintenance of remission, the specific treatment of the central nervous system leukemic sanctuary early in remission and improvements in supportive care have resulted in a current disease-free, complete remission rate of greater than three years for approximately 50% of children with ALL.[1]

Further refinements or improvements in therapy will occur as the result of one or more of several different approaches. First, further investigation into the etiology of leukemia could lead to identification of the cause of this illness and ultimately to the means to prevent it. Second, improved treatment of all groups of acute leukemia might be accomplished by the development of new chemotherapeutic agents or new adjuvant measures such as immunotherapy. Such measures are being tested in ongoing clinical trials. Third, improvement in characterization and classification of subtypes of acute leukemia will lead to recognition of those subtypes which are very responsive to current therapeutic modalities and identification of types resistant to current treatment for which new forms of therapy will need to be developed.

An example of the latter has been the subclassification of ALL in children according to cell surface markers. It had previously been recognized that children with ALL who had clinical features such as age less than 2 years or greater than 10 years, over 50,000/mm^3 circulating lymphoblasts in the peripheral blood or a mediastinal mass tended to have a worse response to therapy than children who did not have these parameters.[2] After T-lymphocytes were identified by their ability to form spontaneous rosettes with sheep red blood cells (E-rosettes) and B-lymphocytes by the presence of surface immunoglobulin (SIg) or by the formation of rosettes with antibody coated sheep red blood cells (EAC rosettes), the malignant lymphoblasts from children with ALL were studied by these techniques.[3,4,5] The majority of children with ALL have lymphoblasts that are predominantly "null cell"; i.e. they have neither T or B surface markers detectable by the methods described above (Table II). Approximately 25% of children with ALL will have the majority of their blast cells form E-rosettes (Figure 1) which indicates that they have T-cell disease and a very small percentage have B-cell leukemia. The clinical importance of these observations is that children with "null cell" disease have a much more favorable response to current therapy regimens as compared to those who have either T-cell or B-cell surface markers.[4] Children with T-cell leukemia also tend to have the clinical features such as a mediastinal mass and elevated white count that are associated with a poor prognosis. It is thus reasonable to treat children with "null cell" ALL with current regimens that are effective in the majority of patients and to direct newer forms of therapy to those children whose ALL is less responsive as determined by clinical presentation and/or on the basis of detectable cell surface markers.

TABLE 1

Type of Leukemia	Percentage of Cases
1. Acute lymphoblastic	80
2. Acute myelogenous	15
3. Acute monoblastic	2
4. Chronic myelogenous	3
5. Chronic lymphocytic	virtually none

Legend: Classification of leukemia in children less than 18 years of age by standard morphological and histochemical criteria.

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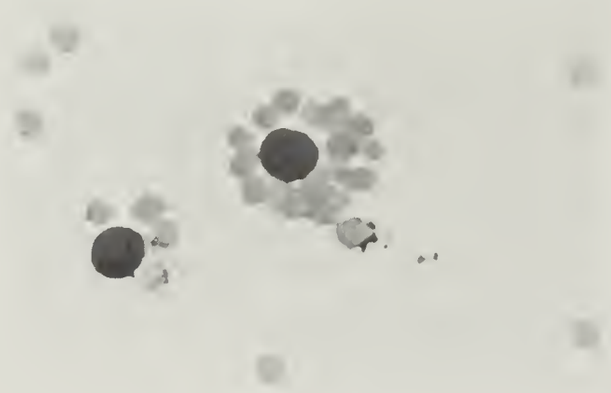


FIGURE 1
T-cell acute lymphoblastic leukemia. The malignant blasts form spontaneous rosettes with sheep red blood cells (E-rosettes).

Another new approach is to classify ALL according to the presence or absence of a biochemical marker. The enzyme terminal deoxynucleotidyl transferase (terminal transferase) is a DNA polymerase which is normally present in the thymus[6] and at lower levels in normal bone marrow.[7] Levels of terminal transferase are not measurable by current methods in normal peripheral blood lymphocytes.[7] However, approximately 95% of children with acute lymphoblastic leukemia have markedly elevated levels of terminal transferase in their circulating blast cells and in the bone marrow during initial presentation and relapse of their disease.[8,9] Elevated levels of terminal transferase are rare in children with acute myelogenous or monoblastic leukemia.

TABLE II
CLASSIFICATION OF CHILDHOOD ALL BY CELL SURFACE MARKERS

Type of Leukemia	Percentage of Cases
1. Null cell (no surface markers)	75
2. T-cell	20-25
3. B-cell	0-5

Terminal transferase has also been found in about 30% of cases of chronic myeologenous leukemia (CML) in blastic crisis.[10] This suggests that CML is a pluripotent disease that may have a lymphoblastic component. Also patients with CML who have detectable terminal transferase are more responsive to chemotherapeutic agents such as vincristine and prednisone which are effective against lymphoblastic malignancies. The assay of terminal transferase is thus useful for both identification of active lymphoblastic malignancies and also for possibly selecting those leukemias which would be responsive to treatment with vincristine and prednisone.

Prednisone responsiveness of ALL has also been determined by the direct measurement of steroid receptors within leukemic cells.[11] Those children with ALL who have a high level of glucocorticoid receptors within their leukemic blast cells have a better response to treatment as compared to those patients who have low levels of glucocorticoid receptors. This biochemical technique also appears to be very promising in its ability to define subclasses of ALL which would be more responsive to therapy.

Newer cytogenetic techniques such as Giemsa banding have also been applied to the study of acute leukemia. Although the banding of bone marrow chromosomes from patients with acute leukemia still poses some technical problems,[12] progress has been made in the area of chromosomal abnormalities associated with acute leukemia. Approximately 50% of patients with acute leukemia have a detectable karyotypic abnormality.[13,14] Although a wide variety of different chromosomal patterns have been noted, the chromosomal changes appear to be non-random in certain specific instances. The classical non-random abnormality is the presence of the Philadelphia chromosome in patients with the adult form of chronic myelogenous leukemia. Recently it has been observed that blasts in acute promyelocytic leukemia have a translocation of the long arms of chromosome 17 to chromosome 15 and also that in acute myelogenous leukemia in adults trisomy 8 is frequently present in the leukemic cell population.[14] Therefore, just as the Philadelphia chromosome has assisted in the classification of chronic myelogenous leukemia, chromosomal changes can also identify certain sub-groups of acute leukemia.

Cytogenetic studies have also provided evidence of the clonal nature of acute leukemia in that the same chromosomal abnormality which is present at the time of initial presentation disappears during remission and reappears when there is relapse of the disease. Studies of chromosomal changes in second malignancies which occur after treatment of the original malignancy will also help to define those malignancies which are new as compared to those which are evolutions of the original cancer. In addition, information as to the effects of cancer treatment with chemotherapy or radiation therapy on specific chromosomes will also be obtained.

Another intriguing speculation as to the role of cytogenetic abnormalities in acute leukemia has been the preliminary observation that the response to therapy in adults with acute myelogenous leukemia is worse in that group of patients who have the greatest percentage of detectable cytogenetically abnormal cells in their bone marrow.[13] Further studies will be required to define the usefulness of cytogenetic determinations in determining the potential response to treatment in children with acute leukemia.

A rare form of acute leukemia, acute monoblastic leukemia, has been better defined by its characteristic histochemical staining pattern.[15] The cytochemical staining patterns of acute lymphoblastic leukemia, acute myelogenous leukemia and acute monoblastic leukemia are outlined in Table III. Use of this cytochemical profile has led to a more accurate classification of acute leukemia.[16] The Periodic Acid Schiff (PAS) reaction may be weakly positive in some cases of acute myelogenous or acute monoblastic leukemia, however acute lympho-



FIGURE 2
Acute monoblastic leukemia. The blast cells show markedly positive staining with alpha naphthol esterase. This stain is a deep red-orange when positive.

TABLE III
CYTOCHEMICAL STAINING OF ACUTE LEUKEMIA

Stain	Lymphoblastic	Myelogenous	Monoblastic
1. PAS	+	±	±
2. Sudan Black B/Peroxidase	-	+	-
3. Specific esterase (chloroacetate esterase)	-	+	-
4. Non-specific esterase (αnaphthol butyrate esterase)	-	+	+

blastic leukemia is characterized by its marked block positivity in the PAS stain along with negative Sudan Black and esterase stains. The pattern of reaction in acute myelogenous leukemia is markedly positive staining with Sudan Black B, peroxidase and chloroacetate (specific) esterase, while the α naphthol butyrate esterase is weakly positive or negative. Due to FDA restrictions on a carcinogenic reagent (benzidine) used in the peroxidase test, we no longer use this stain as identical information can be obtained from the pattern of staining with Sudan Black B.

Blast cells from acute monoblastic leukemia stained with Wright's stain tend to appear relatively uniform in size with diameter of 18 to 25 microns and have abundant blue-gray cytoplasm. Fine azurophilic granulation may be present in the cytoplasm. These blasts are very undifferentiated in appearance and have been confused with both myeloblasts and lymphoblasts. The characteristic cytochemical staining is a strongly positive reaction to α naphthol butyrate (non-specific) esterase in acute monoblastic leukemia (Figure 2 while in contrast to AML the Sudan Black stain tends to be weakly positive or negative. The identification and recognition of acute monoblastic leukemia is important as patients with this disorder tend to have a different response to therapy compared to children with ALL or AML.

Myeloid precursor cells, stimulated by colony stimulating factor (CSF), will form in-vitro colonies (CFU-C) in semisolid media such as agar or methylcellulose. This technique has been applied to the study of bone marrow cells in acute leukemia by several investigators.[17,18,19]

TABLE IV
BONE MARROW TESTS IN THE CLASSIFICATION OF ACUTE LEUKEMIA IN CHILDREN

I. Standard Techniques
 1) Histochemistry
 a) Wright's stain
 b) Special stains (PAS, Sudan Black, esterase)
 2) Surface markers in ALL (E-rosette, EAC rosette)
II. Investigational Techniques
 1) Terminal transferase
 2) Steroid receptors
 3) Cytogenetics
 4) In-vitro colony growth

Technical differences in the culture systems make comparisons between the results of different laboratories very difficult, but one recent study of acute myelogenous leukemia in adults utilizing an agar culture system has defined three groups of AML on the basis of cell growth and colony formation in the in-vitro system. Those patients who had an extensive in-vitro proliferation of colonies and large aggregates had a much worse response to the chemotherapeutic regimen used as compared to patients whose in-vitro colony formation was less extensive.[19] Additional investigations are necessary, including an evaluation of this technique in children, to confirm these findings. The evidence from in-vitro culture experiments such as the above and from studies of the cellular kinetics of acute myelogenous leukemia indicate that AML is not a homogenous entity and that there are certain subtypes of AML which have differing responses to therapy.

SUMMARY:

A bone marrow examination remains necessary for the establishment of the diagnosis of acute leukemia. Advances in morphology and histochemical staining of the leukemic blast cells within the marrow has been helpful in categorizing acute leukemia, particularly in defining the rare type of leukemia now recognized as acute monoblastic leukemia. However the ability to define specific subgroups such as T-cell ALL within the major categories of acute leukemia by morphological and histochemical criteria alone remains limited. Recent investigations into the functional aspects utilizing cell surface markers, biochemical markers and cell culture techniques have been developed as supplements to standard morphology (Table IV). The determination of cell surface markers in children with acute lymphoblastic leukemia to define T-cell disease should be considered standard procedure. Other techniques remain investigational, and two of the authors (JJH and FLM) are continuing to pursue the prognostic significance of terminal transferase determinations in children with leukemia. Cytogenetic analysis of the blast cell population has also provided further information about subgroups of acute leukemia. These new techniques discussed in this review have been useful in defining subgroups of children with ALL who are more responsive to current therapy, and have provided suggestive evidence that acute myelogenous leukemia can also be divided into subgroups with different therapeutic responses.

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Reactions To Radiologic Contrast Media:

When Does An Emergency IVP Take Precedence?

Mark A. Silvert, M.D.

An acutely injured patient with hematuria who coincidentally has a history of contrast material reaction presents a management dilemma that is being seen frequently in the emergency ward. The decision to obtain an IVP or not has grave implications not only for the injured patient, but for the primary physician and radiologist in our modern medico-legal climate. While the radiologist is justifiably concerned with the hazard of the contrast material he is responsible for dispensing, the primary care physician often finds himself in an almost adversary role trying to obtain an adequate evaluation of the injured patient. Obviously, the physicians involved have to make a determination whether the potential value to the patient of the intravenous pyelogram is greater than the risks of readministering the contrast agent. By reviewing the incidence, risk factors, and possible prophylaxis a factual basis will be readily available to aid in this critical decision.

Mechanism of "reactions"

Despite the attention contrast material reactions have received, the mechanism is essentially unknown. Clearly, these reactions are not primarily allergic in nature but are due to a variety of causes including hypertonicity of the media, chemotoxitity, idiosyncrasy, or even secondary to anxiety.[9]

A number of factors make an allergic or immunologic mechanism unlikely. For example, many reactions occur in patients who had not previously been exposed to the media although the patients could have been exposed to the preservatives and chelating agents many contrast materials contain. In addition, repeated injections do not seem to increase the incidence of adverse reactions. Also, the organic iodides chemically are poor haptens. Finally, the evidence of antibodies against contrast media are not

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TABLE I
INCIDENCE OF CONTRAST MATERIAL
MORBIDITY/MORTALITY

	Number of Patients	Study Duration	Procedure	Minor	Moderate	Severe	Deaths
Pendergrass[12] (1958)	11.5 million (est.)	1942-56	IVP				.0086%
Ansell[1] (1970)	318,500	1966-69	IVP		.044%	.0075%	.0025%
Witten[17] (1973)	225,000	1959-74	IVP				.0013%
	32,964	27 mos.	IVP	5.1%	1.63%	.091%	.0030%
Davies[4] (1975)*	3,509	23 mos.	IVP	8.0%	1.5%	0.2%	-
Gooding[8] (1975)*	12,419	3 mos.	Pediatric IVP	5.4%		0.056%	-
Shehadi[14] (1975)	81,278		IVP	3.8%	1.8%	.022%	.0074%
	24,250		Arterial	1.3%	0.8%	.045%	.0124%

* Prospective Study

very strong. Lasser[10] and Dunn[5] were unable to produce antibodies to contrast media experimentally. Brasch,[3] however, has recently reported demonstrating specific IgG and IgE in rabbits as well as demonstrating antibody activity in 27 patients with severe reactions.

Chemotoxic theories have a number of studies to support their hypotheses.[2,10] Pharmacologic activities demonstrated to date include enzyme inhibition, vascular damage, coagulation/fibrinolysis effects, histamine liberation, and complement activation.

Incidence

In the almost fifty years since contrast media were introduced in clinical medicine,[15] information regarding "reactions" has been constantly accumulated. From the literature, it is apparent that the mortality has not changed significantly in over thirty years. Epidemiologic studies have shown that the incidence of toxicity is highest in the 3rd and 4th decades, approximately evenly distributed between the sexes, and occurs somewhat more frequently in those with an "allergic" history.[14]

Morbidity is much harder to quantitate since a variety of classifications have made comparisons of different authors difficult. The spectrum of reactions ranges from a flushed feeling to anaphylaxis. Minor reactions include pain at the injection site, arm cramps, nausea, flushing, and mild rash. These reactions usually do not require medication or delay the radiologic procedure. Moderate reactions often delay the procedure and usually require medication. This category includes extensive urticaria, faintness, rigors, bronchospasm, dyspnea, severe vomitting, headache, abdominal pain, chest pain, and angioneurotic edema.

Severe reactions are life threatening: vascular collapse, loss of consciousness, pulmonary edema, cardiac arrest and cardiac arrhythmia. Not only is the procedure delayed or cancelled, but the patient usually is hospitalized after the acute reaction is stabilized. Since most reactions occur from one to ten minutes post injection, it is important that a physician be readily available during this critical period.

Table I reviews the incidence of contrast material reactions. Ansell[1] stated that 1/3 of intermediate reactions had an allergic history. However, Davies,[4] in a prospective study, could not find a statistical difference regarding an allergic history.

The importance of injection time is also unclear. Ansell[1] was unable to draw any conclusions while Shehadi[14] showed a statistically significant decreased incidence if the injection time was less than two minutes. Davies[4] determined that doubling the contrast material dose also did not affect the incidence of reactions. Although Ansell[1] found a threefold increase, he noted that the patients receiving the higher doses in his series were probably the higher risk patients receiving infusion pyelography. In contrast, Shehadi[14] studied bolus vs. infusion pyelograms and found an insignificant difference in reactions (5.38% and 7.06% respectively).

Pretesting

In an effort to predict who will have a reaction and who will not, a number of predictive tests have been developed. Intradermal, subcutaneous, sublinqual, ocular, oral, and intravenous tests have all been evaluated and discarded when they proved to be ineffective. Only pretesting with a ½ to 1 ml. of intravenous contrast media is still utilized with a significant frequency.

Fischer,[6] after analyzing the deaths caused by 3.8 million pyelograms, was unable to demonstrate any difference in the death rates between radiologists who pretested and those who did not. Witten,[16,17] in a prospective series of 10,000 urograms, failed to predict a reaction in 559 of 569 patients in whom a clinically important reaction developed. Finally, Shehadi[14] found that the incidence of reaction in patients without pretesting and those with negative results was equivalent to that of the general population. Of 375 patients with a positive reaction, only five had their exam cancelled. The remainder had the full dose injected following which 76 had no further reaction, 209 had minor reactions, 87 had moderate reactions, and 3 had severe reactions culminating in death for 2. The readiness of the emergency cart protects the patient far more effectively than the unpredictable and unreliable pretest.

Premedication

If predicting an emergent reaction is unsuccessful, why

TABLE II
INCIDENCE OF CONTRAST MEDIA REACTION AFTER PREVIOUS REACTION

Author		No. in Study	Mild	Moderate	Severe	Comments
Schatz[13]	(1975)	43	16%	11.8%	-	Pretreatment with Benadryl. 50 mg I.V. or P.O.
Shehadi[14]	(1975)	1714		16.2%	-	Previous reaction to same exam.
		204		21.6%	-	Previous reaction to other exam.
Witten[17]	(1975)	66		35.0%	-	Moderate previous reaction. Pretreated with antihistamines.
		55		1.8%	-	Mild previous reaction. ± antihistamine.
Zweiman[18]	(1975)	23	-	-	-	Mild previous reaction.
		46		8.7%		Moderate to severe previous reaction. Pretreated with steroids 18 hours before and 12 hours after exam.

not prevent it with proplylactic medication? Unfortunately, premedication with antihistamines or steroids has not been very useful. Shehadi[14] was unable to draw any conclusions while Ansell,[1] Gates,[7] and Ochsner[11] were able to demonstrate a reduction in minor and moderate reactions only. These reactions could easily have been treated expectantly with antihistaminic and/or steroidal medication when reaction occurred without increased risk to the patient. Heparin also has not been shown to be clinically useful.

Incidence of a Reaction After a Prior Adverse Reaction

If one has had a contrast reaction, what are the probatilities of having a second reaction, perhaps more severe upon reexposure to the media? If contrast reactions were immunologic, one would expect a high recurrence rate. Fortunately, this does not seem to be the case. As shown in Table II, there is an increase in the mild and moderate reactions despite all of these studies being pretreated with a variety of pharmacologic agents. However, the incidence is only double to triple that of the unexposed population. Significantly, in these studies, the severe and death rates were not increased. Thus, we can conclude that with adequate preparation, the reinjection morbidity and mortality rate is not significantly raised enough to defer a justifiable IVP.

Summary

Contrast media reactions continue to be a perplexing and potentially dangerous problem. Few reactions seem to be truly allergic. Rather, the effects are primarily chemotoxic, idiosyncratic and secondary to anxiety. Pretesting, premedication, and obtaining an allergic history are of minimal value in preventing reactions.

The patient with a history of a previous reaction should not be deprived of a medically indicated pyelogram. A good history of the previous reaction is mandatory to determine the severity of the previous reaction. If only a history of a mild vasomotor character can be extracted, the exam can be done without a significantly increased risk. A moderate to severe reaction would indicate that the repeat exam should be performed with appropriate premedication (antihistamines or steroids). Although one can expect a 2 to 4 fold increase in moderate reactions, treatment of these reactions should not be difficult with appropriate preparation. In the case of a previous severe reaction, anesthetic standby and intravenous access as well as premedication is strongly recommended. For possible severe renal trauma, one should consider renal arteriography as the initial procedure since not only is it difinitive, but may have a lower incidence of reactions.

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"Timely" Diagnosis Of Viral Infections

C. George Ray, M.D.

Viruses are important and common causes of morbidity in our society, yet they are regarded by many as possessing a certain mistique, as well. The situation is somewhat analogous to the "veiled ladies" of the middle East, where one wonders what is behind the veil, yet hesitates to find out, either out of fear of reprisal, the possibility that one may be disappointed at the view, or merely the fact that any attempt at unveiling would be unsuccessful, anyway. The following is an effort to strip the viruses of some, but not all, of this mystique, and to place viral diagnosis into a perspective which can be useful to the clinician.

In humans, viruses can behave in a variety of ways, some of which have only recently been recognized. These range from asymptomatic infection or acute clinical disease (the most common forms observed), to sophisticated methods involving integration into the host genetic code with permanent alteration of cell function. Table 1 illustrates some examples of these behavioral modes; however, it should be pointed out that, while highly probable, human counterparts to lower animal cancer and autoimmune disease-associated viruses have not yet been conclusively proved. One aspect of virus behavior that is of paramount concern to the clinician is their frequent uncanny ability to produce illnesses which closely mimic other diseases, such as bacterial or mycotic infections, blood dyscrasias, or autoimmune-like phenomena.

Viruses are not only ubiquitous in terms of morbidity, but also with respect to the diversity of types available to afflict. There are approximately 240 different serotypes currently recognized as important human pathogens, arranged into about 19 groups, based on chemical, physical, and biological properties or their ecological distribution in the host or in nature. Despite this large number, it is now possible to offer specific laboratory diagnosis for the majority.

Traditionally, there have been several important objections to the development of diagnostic virology laboratory services that can be summarized as follows:

1. **The results are too slow to benefit the patient or clinician.** This is clearly no longer the case. Most infections can be diagnosed and a preliminary report issued to the physician within a few days. Our own experience indicates that preliminary results are available within five days in approximately 65% of proved infections, and, with the increased use of rapid diagnostic methods (to be discussed later), many results can be reported within 3 to 24 hours.
2. **The methods of diagnosis are too insensitive, resulting in a low diagnostic yield.** In the past, many laboratories cited an overall diagnostic yield of 5% to 10% of all patients studied. With improved methodology, this yield has risen to 30% to 40%, and includes patient studies where negative results were anticipated, e.g., to "rule out" a specific viral infection.

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TABLE 1
VIRAL OPTIONS IN THE HUMAN HOST

	Examples
1. Infection, no disease	Any virus
2. Infection, acute illness	Influenza, Measles, Coxsackie viruses, etc.
3. Infection, with or without acute illness, followed by chronic persistence in the host.	
(a) Latent, inactive, with ability to reactivate and produce acute disease	Varicella-Zoster virus ("shingles"), Herpes simplex virus ("cold sore")
(b) Chronic, productive, with progressive host damage	Hepatitis B virus (chronic active hepatitis), "Slow virus" diseases of the central nervous system, "?Autoimmune" diseases.
(c) Integration of viral genome into the host cell, altering cell behavior	?Cancer viruses

3. **Since there is no specific treatment available for viral infections, why bother to diagnose them?** This is only partly true at the present time. While widespread, effective chemotherapy for viral infections is primarily a hope and expectation for the future, such therapeutic possibilities are already beginning to materialize. For example, adenine arabinoside for systemic treatment of severe herpes simplex infections, and amantadine HCl for the prophylaxis and possible treatment of influenza A infections have shown some promising results, and other agents are currently being tested for efficacy. The experience to date has indicated that specific therapy will have to be based upon a knowledge of the pharmacology of the antiviral agents as well as a precise diagnosis as to which viral agent may be involved in the illness, a situation analogous to that which is faced now with regard to antibacterial therapy. For the present, however, the major therapeutic implications of a positive viral diagnosis are nihilistic, in that it can lead to a clearer understanding of the disease process and greater precision regarding prognosis. This can often result in discontinuation of unnecessary antibiotic therapy and further laboratory tests, and sometimes also leads to a shortened duration of hospitalization.

TABLE 2
WHY DIAGNOSTIC VIROLOGY?

1. Epidemiologic monitoring.
2. Physician and patient education.
3. Definition of the disease process and its prognosis.
4. Therapeutic implications (positive and negative).

The major reasons for diagnostic virology to exist are summarized in Table 2. Some, such as epidemiologic considerations, continue to be extremely important to the community at-large, and necessary for public health decision-making. It has been only in recent years that the possibility of direct patient benefit has become recognized. Table 3 is a summary of the major specific situations where viral diagnosis has proved to be of particular value. There are also other syndromes, including polymyositis, blood dyscrasia-like or auto-immune-like illnesses, and unusual genitourinary tract diseases, where viral studies have yielded interesting and sometimes surprising results. These often raise important and intriguing questions regarding the role of viruses in a wide variety of human diseases, but a "shotgun" viral diagnostic approach to unusual illnesses must still be tempered with reasonable forethought as to whether the results will truly benefit the patient. Similarly, it is not in the best interests of medical care to insist upon culturing every patient with a presumed viral infection, particularly if the illness is mild.

TABLE 3
SITUATIONS WHERE VIRAL DIAGNOSIS CAN BE HELPFUL

	Agents Frequently Found
Febrile Central Nervous System Syndromes	
"Partially treated" meningitis, "Aseptic" meningitis, Encephalitis, Transverse myelitis, Polyneuritis, etc.	Enteroviruses, Mumps, Herpes simplex; Occasionally Arboviruses, Cytomegalovirus, Epstein-Barr virus
Outbreaks of Lower Respiratory Disease	Influenza A and B, (all ages); Respiratory Syncytial virus, Parainfluenza viruses (infants and children); Adenoviruses
Severe Pneumonia, Unresponsive to Antibiotics, and No Bacterial Etiology Determined	Influenza A, Adenoviruses
Myopericarditis, Acute Myocardiopathies	Coxsackie B viruses
Acute Gastroenteritis	Rotaviruses (especially infants and children)
Exanthems and Enanthems	
Macular and Maculopapular	Rubella, Measles, Enteroviruses, Adenoviruses
Vesicular	Herpes simplex, Varicella-Zoster
Congenital or Perinatally Acquired Illness	Cytomegalovirus, Herpes simplex, Rubella, Enteroviruses
Febrile, Mononucleosis-like or Sepsis-like Syndromes	Epstein-Barr virus, Cytomegalovirus, Adenoviruses, Enteroviruses
Conjunctivitis, Keratitis	Adenoviruses, Herpes simplex,
Hepatitis	Hepatitis; A, B, or non-A, non-B; Cytomegalovirus, Epstein-Barr virus, Adenoviruses, Coxsackie B viruses

HOW ARE VIRAL DIAGNOSES MADE?

There are many methods available to a virology laboratory to establish a diagnosis. Because of the large number of viral agents which can cause infection, and the diverse methods which are necessary to detect each of them, it is absolutely essential that the physician communicate with the laboratory *before* ordering cultures.* This is underscored by our experience during the 1977 calendar year, where 51% of illnesses which were studied after appropriate communication with the ordering physician were specifically diagnosed, as compared to only 24% when communication was not obtained

TABLE 4
APPROPRIATE SPECIMENS FOR VIRUS ISOLATION*

AGENTS GENERALLY SOUGHT	SPECIMENS					
	Throat	Stool	CSF	Urine	Vesicle*** Fluid	Other
Meningitis-Encephalitis						
Mumps	++++	--	++	+	--	--
Enteroviruses	+++	++++	+	--	--	--
Herpes Simplex	±	--	±	--	+	Brain Biopsy ++++
Arboviruses**	--	--	+	--	--	Brain ++ Blood+
Respiratory Diseases						
Influenza, Parainfluenza viruses	++++	--	--	--	--	
Rhinoviruses Adenoviruses, Enteroviruses	++++	++++	--	--	--	
Exanthems						
Rubeola** (Measles), Rubella**	++++	--	--	+	--	
Variola***	++	--	±	±	++++	
Vaccinia***	--	--	--	--	++++	
Varicella***	--	--	--	--	++++	
Herpes simplex***	++	--	--	--	++++	
Enteroviruses	+++	++++	--	--	+	
Myocarditis-Pericarditis						
Enteroviruses**	++	+++	--	--	--	Pericardial fluid++
Influenza viruses**	+++	--	--	--	--	
Other						
Cytomegalovirus	++	--	--	++++	--	Leukocytes +

* In general, it is important to remember that virus shedding often diminishes rapidly after the onset of illness; therefore, it is inportant to attempt to collect specimens as early as possible including an acute serum for future testing. (-- = no yield; ± to ++++ = relative yield)

** Because it is frequently very difficult to isolate and/or associate these agents with the disease in question, it is emphasized that serological tests are particularly important in order to insure a diagnosis.

*** Do not prep skin with alcohol or iodine before obtaining samples.

beforehand. The diagnostic yield was not influenced to any degree by distance; positive results were possible when patients living more than 150 miles from our laboratory were studied, as long as communication, proper specimen collection, and reasonable transport procedures were followed.

Once a clinical history is obtained, the possible viral agents involved and the proper specimens necessary for diagnosis can be determined. Table 4 summarizes some of the major agents according to syndrome, and the relative diagnostic yield one can expect from different sampling sites.

The most common culture method in the laboratory is by the use of tissue culture monolayers. A variety of cell types are utilized, depending upon the viruses being sought. Specimens are processed and inoculated into the cell cultures, and observed daily for evidence of virus replication, which can be manifested by alteration of cell morphology or cell destruction (cytopathic effect), or, in some instances, by inducing the cells to adsorb red blood cells to their surfaces (hemadsorption). In some circumstances (e.g., rubella) the virus induces neither cytopathic effect or hemadsorption; its presence is detected by the ability of the cell sheet to resist infection when later challenged with a second virus which normally will produce a cytopathic effect (interference method). Some viral agents require inoculation of newborn mice or embryonated hen's eggs in order to be detected, and still others, such as rotaviruses, will not grow at all in routine culture systems, and are usually visualized by direct examination of diarrheal stools by electron microscopy (Figure 1).



FIGURE 1
Rotavirus particles in diarrheal stool visualized by direct electron microscopy. Original magnification, X60,000 (Courtesy, Dr. Claire Payne).

The culture methods are often reasonably sensitive, yielding evidence of a virus within 1 to 14 days, depending upon the type involved, and the amount present in the original inoculum.

As mentioned earlier, other methods have evolved, and are continuing to be developed, which allow rapid diagnosis within a few hours, in some instances. These include direct electron microscopy, immune electron microscopy, fluorescent antibody techniques, and others, all designed to detect virus particles or viral antigens in body fluids, exfoliated cells, or biopsy materials. One example is the ability to diagnose herpes simplex infection in brain biopsy material within a few hours after surgery by direct immunofluorescence, thus rapidly establishing a disgnosis, effectively ruling out other considerations, and influencing decisions regarding possible antiviral chemotherapy.

Finally, serological methods should not be forgotten. They remain as valuable tools in viral diagnosis, particularly when attempts at culture are unrewarding, and a viral infection is still strongly suspected. In fact, some viral diseases remain in which serological diagnosis is the most sensitive, and sometimes most rapid, procedure available. Examples of these include rubella and some arbovirus infections. It is our policy to recommend collection of acute sera as early in the illness as possible, with the possibility that a convalescent sample may be desirable in two to three weeks. Serodiagnosis of viral infections is most reliable when acute and convalescent serum samples can be tested simultaneously, to determine whether a significant (four-fold or greater) titer change has occurred.

CONCLUSION

Laboratory diagnosis of viral infections has become a service which can be useful to physicians. If utilized properly, such studies can be of considerable direct benefit to patients. However, this is not meant to imply that physicians who can not use all aspects of such services are placing themselves or their patients at a serious disadvantage. The present state of the art is such that, except in some special situations, the benefits are measured primarily as greater diagnostic precision and resultant less uncertainty regarding the diagnosis, work-up, and management of patients with suspected viral disease.

ACKNOWLEDGEMENT

Linda L. Minnich, M.S., gave valuable assistance in the development of data included in this manuscript.

* The Arizona Health Sciences Center Virology Laboratory can be contacted at (602) 882-7745 weekdays, and at other times by calling (602) 882-6300 and asking the operator to page the person on-call for Clinical Virology.

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Evaluation Of The Patient With Vertigo

Charles F. Koopmann, Jr., M.D.

"Dizziness" is a complaint frequently encountered in the practice of many primary care practitioners. The evaluation of this problem can be difficult, frequently resulting in failure to find a specific, organic etiology. However, despite the frequent frustrations associated with the work-up of the "dizzy patient," there are certain steps which, when followed, can make the investigation much easier for the clinician. Thus, this article is designed to present a systematic approach to the evaluation of the dizzy patient without dwelling on specific clinical entities and their treatment.

The first, and most important, step in the management of dizziness is the history. Without an accurate, detailed history, the physician may proceed in the wrong direction and order unnecessary, expensive tests. Consequently, one should ask the patient to describe his spells *without* using the word dizzy. Does he mean that the room spins (object vertigo), that he spins (subjective vertigo), or is the sensation merely faintness or unsteadiness? Is the dizziness affected by body or head movements or does it come on at anytime (i.e. when he is sitting still or lying down?); are there other concurrent symptoms such as a prodromal tinnitius, fullness in one or both ears, decreased hearing, nausea, or vomiting (frequently seen in Meniere's Disease)? Does the spell last for only seconds or does it last for hours or days? How frequently do the attacks occur? Does the patient have a history of previous ear surgery or drainage from one ear (possibly a fistula of the semi-circular canal)? Does he have a history of other concomitant medical problems such as cardiac arrhythmia, hypertension, diabetes, or other endocrine abnormalities, recent viral infections (i.e. a URI might suggest viral labyrinthitis), venereal disease, recent head trauma of the flexion-extension variety, or increased stress at work, school, or home? Are there associated visual symptoms (i.e. diplopia or scotomata), paresthesias, weakness of extremities, dysarthria, or loss of consciousness? Finally, a careful evaluation of all medications is essential since many may cause orthostatic hypotension or, in the case of birth control pills and ethanol, neurological sequelae.

Next, the practitioner must perform a comprehensive physical examination. One should check the cardiovas-

From: University of Arizona Health Sciences Center, Tucson, AZ 85724.

cular system for evidence of hypertension, orthostatic hypotension, cardiac arrhythmias, or carotid bruits. The fundi may reveal papilledema. Examination of the ears should include careful observation for evidence of chronic suppurative otitis media (i.e. purulence in the external auditory canal, cholesteatoma, tympanic membrane perforation). If the examiner has access to a pneumatic otoscope, he can determine if a labyrinthine fistula exists by application of positive pressure to the middle ear space or tympanic membrane. If the patient's eyes deviate to the contralateral side during application of the positive pressure, then the fistula test is considered positive and it is assumed that a labyrinthine fistula may be present. Please note that this test is objective and one should not be influenced by the patient who says he gets dizzy during the test since dizziness without deviation of the eyes to the contralateral side is not uncommon and *is not* a positive test.

Neurological testing is very important. In the head and neck region special attention must be placed on evidence of VII nerve weakness, V nerve anesthesia or paresthesia, evidence of oral or hypopharyngeal anesthesia, evidence of vocal cord weakness, and evidence of anesthesia in the external auditory canal (Hitzelburger's Sign). Also, one should include evaluation of past pointing, rapid alternating movement, tandem gait, Romberg Sign, and intact vibratory, pin prick, and vibrating sensation. Finally, the patient's eyes may reveal gross nystagmus (horizontal, vertical or rotary).

After a thorough history and physical examination, the clinician is presented with the task of initiating an orderly search in confirmation of his clinical suspicions. Anemia, hypoglycemia (diabetic or nondiabetic), and hypothyroidism may be manifested by balance disturbances and may be found by a CBC, glucose tolerance test (5 hour), and T_4 respectively. Recently, syphilis has been associated with hearing loss, tinnitus, and vertigo, probably due to both endolymphatic hydrops and osteitis of the otic capsule. The standard VDRL may be nonreactive but usually the FTA-ABS is reactive. When the hearing loss is sudden and severe, some authorities feel a true medical emergency exists and begin treatment of steroids (Prednisone 40mg/day) and penicillin 20 million units daily for seven days.

Audiological testing is of immense value in the evaluation of vertigo. Pure tone audiometry will show whether a conductive loss (possibly secondary to otosclerosis, cholesteatoma, or post-traumatic ossicular discontinuity) or a sensorineural loss (Meniere's, syphilis, acoustic neuroma) exists. Speech audiometry, as manifested by the speech reception threshold (SRT) and speech discrimination are of assistance in determining whether a lesion may be cochlear, (relatively good agreement between pure tone average and SRT) or retrocochlear (elevated SRT with poor discrimination). Recruitment (defined as an abnormal loudness growth as intensity of sound is increased) can be evaluated by the alternate binaural loudness balance (ABLB) test and helps pinpoint the site of lesion to the Organ of Corti (more specifically to the hair cells) and thus, to cochlear impairment. The SISI test (short increment sensitivity index) measures the ability of the recruiting ear to detect small (ldB) increments of intensity rather than the average 3dB increments usually seen in the normal ear. Tone decay (adaptation of loudness in response to a continuous tone) can be pathogenic of any lesion of the eighth nerve and must be evaluated in conjunction with other hearing tests.

Most recently it has been shown that a relatively new test, the stapedius reflex, is the most common abnormal finding in eighth nerve tumors. The stapedius muscle contracts at levels 70-80dB above threshold to protect the inner ear from loud noise. This reflex is bilaterally symmetrical (mediated in the pons) and thus, sound stimulation of one ear produces bilateral stapedius contraction, easily measured by the impedance bridge. A 20-30dB rise in threshold secondary to a conductive loss elevates the threshold so that the reflex is not noticed. However, if there is no conductive component, the stapedius reflex is a powerful aid in distinguishing cochlear vs retrocochlear lesions.

Evaluation of the vestibular portion of cranial nerve VIII may be done with office ice water calorics or the more sophisticated and accurate electronystagmography (ENG). Caloric testing can be semi-reclining (head elevated approximately 30 degrees) and the water injected against the tympanic membrane at a rate of approximately 1cc/sec. The latency (time from injection to onset of nystagmus) and the duration of nystagmus is evaluated as well as the number of beats per unit of time. However, not only is it difficult to quantitate the above parameters, but it is impossible to prevent suppression of nystagmus secondary to ocular fixation, even with Frenzel glasses. Thus, the ENG can offer the practitioner: 1) evaluation of spontaneous nystagmus 2) evaluation of latent nystagmus (nystagmus with the eyes closed) 3) give permanent recordings (tracings comparable to EKG's for serial evaluation and reference) 4) obtain an ocular fixation index (defined as the number of beats per second with eyes open/number of beats per second with eyes closed). The ocular fixation index is important in that a peripheral lesion usually has a value of one or less while a central lesion has an index of greater than one.

Radiographic examination of the temporal bone is of assistance if an acoustic neuroma is suspected. Standard skull x-rays are useful if a lesion is quite advanced but are of little value in smaller lesions. The Stenver, Towne, and Caldwell views are helpful as screening studies when one is searching for erosion of the internal auditory canal (IAC). However, for small lesions of 1-3 mm, hypocycloidal polytomography of the IAC's gives much more information. Even using the latter radiographic studies, there may be a 10% incidence of false negative studies. Brain scans are useful in the evaluation of large posterior fossa lesions and in coexistent supratentorial lesions but are of little value in small IAC tumors. Computerized tomography (CAT Scan) is a highly sophisticated,

non-invasive method of evaluating intra-cranial pathology. It is useful in patients with large posterior fossa lesions but to date many investigators feel it may fail to show lesions less than 1.5 cm in diameter. Most authors feel that the CAT Scan should be used only after polytomography. It can be used safely in patients with increased intra-cranial pressure and may, if positive, obviate the need for posterior fossa myelograms.

After one has evaluated the patient with the above non-invasive methods, there may remain questions concerning the presence, size, or vascularity of a lesion. Consequently, one now must consider invasive measures. Spinal fluid protein analysis may be utilized in patients without increased intra-cranial pressure. In larger tumors, the protein level should be elevated. However, because of a 25% false negative incidence, spinal fluid protein is used only in conjunction with myelography.

In many centers, a posterior fossa myelogram has been considered the definitive study for confirmation of a cerebello-pontine angle mass. Many people feel that it is the only technique capable of demonstrating the 2.5% acousitic neuromas which are entirely intra-canalicular. The incidence of false positive tests is 5% due to arachnoid adhesions or cyst formation. Cerebral arteriography is useful if one wishes to evaluate the carotid or vertebro-basilar system or if it is necessary to determine tumor size after an abnormal CT scan or myelogram.

Finally, in cases of uncontrollable vertigo due to suspected endolymphatic hydrops, some authors feel that a perilymph protein determination (via a trans-stapedial puncture) is advisable to rule out an unsuspected acoustic neuroma. Marked elevation of the perilymph protein may be present despite normal CSF protein values.

In summary, the common complaint of dizziness can result from a large number of etiological causes. After a careful history, the screening test battery of: 1) a neurotologic examination 2) caloric test 3) audiogram 4) stapedius reflex test 5) routine IAC x-rays will assist the clinician in determining which, if any, of the more sophisticated, expensive diagnostic measures is warranted.

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Arizona Medicine

Have We Been Successful?

As the year draws to a close one usually likes to reflect upon the progress that is made during the preceding months. At ARIZONA MEDICINE a number of significant alterations have occurred and it is indeed rewarding to receive mail praising the journal. However, the number of people responding in such a fashion is small and the negative comments are usually made privately. We have tried to make ARIZONA MEDICINE, as noted in a previous editorial, more conducive to the general physician attempting to keep abreast of the latest advancements in various fields. The journal as a whole I believe has fulfilled this function. There are no. doubt exceptions to this general consensus but on the whole I think the Editorial Board has succeeded. Special mention should be made of the outside reviewers who have given their time freely to help in the selection process for the journal. Many of these people go unrecognized and it is the object of the editorial to praise them. It is very gratifying to see practicing physicians take an active interest in keeping their fellow members abreast of recent developments in their fields. The involvement of the University in this continuing education process is of course most important and appreciated. As Editor, I would like to encourage all physicians throughout the state to contribute to their state journal. Being in Phoenix I am limited in knowing who to ask to review articles that are submitted and who to invite to write articles for publication. The Editorial Board needs the names of those people who would be willing to fulfill these functions for us. I especially want to encourage physicians who have unique contributions to submit them to us, even if it is in an informal fashion. The words themselves, can always be changed, but it is the thoughts that are the important ingredients. I believe there are physicians in areas in the state who have experiences which for us "city folk" would be very enlightening. We need to tap that reservoir.

This editorial, then, is "a thank you" to those who have helped in the past and to encourage those, who may be a bit reticent, to help in the future. The strength and future of this journal is dependent upon its readership.

Marshall B. Block, M.D.
Editor

From The Dean

It is an honor for the College of Medicine to be invited by the Editorial Board of *Arizona Medicine* to provide the scientific papers for this issue. Because faculty of the College owe so much to the citizenry of Arizona, we are not only obliged but pleased to share the wealth of medical knowledge, old and new, well established and recently acquired.

The College of Medicine has been most fortunate in attracting superb scholars to its faculty. They have come from within the borders of Arizona and from distant states, and all have been welcomed not only through the generous provision of a fine physical facility, but through the loyal support and encouragement of a strong organization of the state's physicians. Arizona's physicians exemplify their attitude toward the need for continued learning through the support of the fine state journal.

The College faculty's stature can be measured in several ways. One measure is the quality of the student body choosing to study with them. By national standards, the entering classes of recent years have been outstanding. The 3.63 grade point average for the class entering in 1977 compares to the 3.47 national mean. The overall Medical College Admission Test (MCAT) score of 609 for the 1977 entering class compares to the 593 national mean. While the performance of these students reflects the quality of their premedical educational background, their preference for the University of Arizona College of Medicine reflects the quality of the College.

Peer review is also a measure of faculty quality. The national competition for limited research dollars is keen. In this competition the College's faculty this past year, received more than $9 million of research funding through federal grants and contracts. Additional monies have been invested in the faculty through projects sponsored by voluntary health agencies, pharmaceutical firms, and private donors.

The faculty's success in achieving its educational and research mission can be measured also in its publications. During the past year over 450 manuscripts were published in nationally recognized scientific journals. Each journal article was accepted only after national and often international peer review. Further, many of the faculty are editors or associate

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editors of scientific journals. One major journal has its editorial offices at the College, and virtually every professor is on at least one prestigious journal's editorial board.

Another index of faculty member's distinction is their selection to chair international symposia. Each year several College of Medicine faculty are awarded this honor. In addition, this past year two international symposia were held in Tucson because of the renown and effort of local faculty members.

Basic science doctoral degree programs and medical and subspecialty programs are growing rapidly. While these programs must sustain the critical eye of peer review for approval and accreditation, the final measure is in students choice to participate in them. Each year, more and more fellows, graduate students, and house officers choose the College of Medicine for their education.

The College of Medicine is the state's resource. As the Dean of its faculty I am very proud, and take great pleasure in sharing their efforts through this issue of *Arizona Medicine*.

Louis J. Kettel, M.D.
Dean

AUXILIARY HIGHLIGHTS

Five of the six organized County Auxiliaries publish newsletters which tie the knots of communication among members. Information lifted from some year-end issues weaves the ambitious pattern of action followed by the Auxiliary.

Maricopa Auxiliary, with 1182 members, used their MEDI-CALL to report: the Breast Self Examination program has been filmed to air on Channel 10 as an educational and community service. Other projects include BSE program in Spanish, the Nutrition for the Elderly program; and the Catastrophe Cottage model continues to teach safety. At the Christmas coffee, a check for $3,100 was presented to Project Mobility which transports quadreplegics around the valley. The Crisis Nursery received formula, baby clothes, and toys; and the historic Rosson House in Phoenix was presented with a check to complete the doctor's office as it would have appeared at the turn of the century. The sale of Holiday Greeting Cards netted $9,000 for the medical school.

Pima Auxiliary, 391 members, reported via LA CARTA: Besides hosting the 1978 ArMA Auxiliary Convention in April, the auxiliary donated $150 to the Crisis Nursery for a new electronic thermometer and sponsored the third Women Aware Conference in Tucson. Attended by 285 women, the Conference featured speaker Eleanor McGovern and offered three classes in various relevant subjects. The Nutrition Skit was dramatized for elementary school children, and a Grand Auction was held in March toward raising$14,500 for the Community Food Bank. The BSE program is being prepared for fall presentation.

Coconino Auxiliary, 33 members, reports through the NORTHLAND NEWS: A very successful Christmas Bazaar netted $3,000 which provided $200 for A.M.A. Education and Research Fund with the remainder being divided between the new Crisis Nursery and the Sunshine Rescue Mission. Their February meeting was held at the Snow Bowl with children, sleds, and skis. Luncheon meetings included one held over the hill in Sedona in addition to the Octoberfest held there in Yavapai.

The YAVAPAI YODLER informs 39 members of Yavapai Auxiliary meeting plans and programs such as the December get-together with husbands for wine-tasting and the gathering of gifts sent with the Christmas Eve Meals for the Elderly. Their annual rummage sale netted $1,000; and monthly bake sales at the hospital, jewelry sales, and the raffle of a special afghan handcrafted by Verna Hough, raised funds to support A.M.A.E.R.F. and refurbish hospital labor rooms. The group cooperates with Kiwanians in sponsoring community Bloodmobile visits.

Yuma Auxiliary published THE BETTER HALF to inform its 46 members of rummage sale profits and $460 raised for A.M.A.E.R.F. through Holiday Sharing Christmas Cards. The nutrition project Muppets were trouped to classrooms, and the Coronary Pulmonary Rescusitation classes held twice weekly resulted in over 250 citizens being certified in basic CPR rescue.

Mohave Auxiliary's 16 members kept busy without benefit of a newsletter and concentrated efforts toward BSE in Spanish and English, amblopia screening, and immunization aimed at pre-school and kindergarten children. A.M.A.E.R.F. was supported by Holiday Greeting Cards and a progressive dinner in Lake Havasu City. Their bi-monthly meetings featured programs on CPR, BSE, eye screening, and child abuse.

Adding members at large, a total of 1418 wives of Arizona physicians carried out a patchwork of activity during 1977-78, and the combined effort provided a king sized blanket of health-related accomplishment over the state.

Mrs. Melvin W. (Jean) Phillips
Auxiliary Editor

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

PHYSICIAN REHABILITATION COMMITTEE

The meeting of the Physician Rehabilitation Committee of the Arizona Medical Association, Inc., held at 810 W. Bethany Home Road, Phoenix, Arizona on Saturday, January 28, 1978, convened at 1:43 p.m., Thomas E. Bittker, M.D., Chairman, presiding.

CASE REVIEWS

The committee reviewed several cases and made recommendations.

YUMA REPRESENTATIVE

It was recommended that George H. Darwin, Jr., M.D. would be a good representative of the committee in the Yuma area.

REPRINT LIBRARY

It was agreed that Mr. Robinson would set up a reprint library as a repository of reprints of articles that members of the committee may wish to place therein.

ANNUAL MEETING - BREAKFAST PANEL

Dr. Bittker outlined the plans for the panel presentation "The Distressed Physician: Where Do We Go From Here?" scheduled for 7 a.m., Thursday, April 27, 1978 in the Ballroom Salon #1 of the Marriott Hotel in Tucson. All committee members were urged to attend.

It was recommended that an invitation be extended to hospital administrators to attend this presentation.

CONTACT TECHNIQUES

The committee reviewed the video tapes on the subject matter provided by the Washington State Medical Association as well as the audio tapes prepared by the chairman as part of the training program for the committee.

NEXT MEETING

It was determined to hold the next meeting on Wednesday, April 26, 1978 at 7 p.m. at the Marriott Hotel in Tucson with Emanuel M. Steindler, Executive Director, Department of Mental Health of the American Medical Association, present.

MEDICAL EDUCATION COMMITTEE

Meeting of the Medical Education Committee of the Arizona Medical Association, Inc., held Thursday, February 9, 1978, at 810 W. Bethany Home Road, Phoenix, convened at 7:00 p.m., Robert E.T. Stark, M.D., Chairman, presiding.

NATIONAL COUNCIL OF STATE CHAIRMEN OF CONTINUING MEDICAL EDUCATION

Dr. Stark reviewed correspondence, proposed by-laws and minutes of the December organizational meeting of the above group. The committee reaffirmed its support of the Council, and will recommend to the Board of Directors that the Arizona Medical Association participate as a member, including payment of the $200.00 annual assessment to member societies.

ArMA CERTIFICATE IN CME

Letter of 11/22/77 from AMA

The above letter from Clarke Mangun, M.D., was reviewed. Criticisms of the ArMA criteria for Category I were discussed. It was felt that many of the differences between the ArMA and PRA criteria can be easily resolved because of ArMA's recent approval as an accredited organization. Several other criticisms, for example, ArMA's approval of preparation for specialty board certification or recertification, education for advanced degree, and a provision for consideration of "special circumstances", need further study and discussion.

ArMA Audit of CME Applicatlons

Dr. Stark proposed that ArMA staff do all auditing of applications for the ArMA CME Certificate, and discontinue the practice of sending them to the AMA for aproval. Staff is presently auditing the applications and mailing to AMA, who accept our audit in Category 1, but are continuing to audit other categories. However, they almost without exception approve the applications we send them and issue the PRA.

IT WAS MOVED AND CARRIED TO APPROVE CME APPLICATIONS LOCALLY AND DISCONTINUE THE PRACTICE OF FORWARDING THEM TO AMA FOR APPROVAL.

A mechanism for informing AMA of those who have received the ArMA Certificate will have to be worked out, and it is hoped that AMA/PRA will continue to reciprocate with the ArMA Certificate.

BOMEX Rules and Regulatlons on CME

The Rules and Regulations adopted by the Board of Medical Examiners on 12/9/77 were reviewed. The committee found several inaccuracies which it was felt should be corrected. It was suggested that a meeting be sought with the BOMEX CME Committee to discuss the rules and regulations.

IT WAS MOVED AND CARRIED TO DIRECT STAFF TO ARRANGE A MEETING WITH THE CONTINUING MEDICAL EDUCATION COMMITTEE OF THE BOARD OF MEDICAL EXAMINERS TO DISCUSS THE RULES AND REGULATIONS ON CONTINUING MEDICAL EDUCATION RECENTLY PROMULGATED BY THEM.

S.B. 1132

Copy of a bill introduced into the Arizona Legislature which would rescind all mandatory requirements for continuing education from Arizona Statutes was received for information.

SECTION REPORTS

Section on Accreditation

A. Dr. Sheeley reported that the Section on Accreditation recommends approval of the reports of the survey teams who resurveyed St. Joseph's Hospital & Medical Center and the Arizona State Hospital. Recommendations are as follows:

1) That St. Joseph's Hospital and Medical Center's continuing medical education program be accredited for a period of four years.

2) That the Arizona State Hospital be granted probationary accreditation of their continuing medical education program for a period of one year, with re-survey at that time; further, that only the Psychiatric Grand Rounds, Medical Grand Rounds, Clinical Pathological Conferences, and Quarterly Drug Therapy Seminars receive Category 1 credit toward the ArMA Certificate and the Physician's Recognition Award. Resurvey team should include two Psychiatric physicians.

IT WAS MOVED AND CARRIED TO APPROVE THE RECOMMENDATIONS OF THE SECTION ON ACCREDITATION REGARDING ST. JOSEPH'S HOSPITAL AND MEDICAL CENTER AND THE ARIZONA STATE HOSPITAL.

B. **Proposed Revlsed Accreditation Materials**

Dr. Sheeley reported that the Section has approved revised accreditation materials submitted by Dr. Hastings, with editorial changes noted.

The Committee members felt they could not make a decision regarding the proposed materials without having reviewed them.

IT WAS MOVED AND CARRIED TO TABLE CONSIDERATION OF THE PROPOSED REVISED ACCREDITATION MATERIALS.

IT WAS MOVED AND CARRIED THAT THE PROPOSED REVISED ACCREDITATION MATERIALS BE CIRCULARIZED BY MAIL TO ALL COMMITTEE MEMBERS, AND THAT THE MEMBERS BE ASKED TO VOTE ON APPROVAL BY MAIL, AND TO SUBMIT SPECIFIC REVISIONS IF NOT ACCEPTED IN TOTO. REPLIES TO BE SENT WITHIN TWO WEEKS OF RECEIPT OF THE MATERIALS.

Section on Certification

A. Dr. Taylor reported that the Section on Certification had reviewed sixteen requests for extension of reporting period or some other type of special consideration in meeting their CME requirements, and that the Section recommends approval in all but one case.

IT WAS MOVED AND CARRIED TO APPROVE THE RECOMMENDATIONS OF THE SECTION ON CERTIFICATION.

B. **Status of 1977 CME**

Dr. Taylor reported that there are as of this date 128 members who have not as yet responded who were due to report their CME by December 31, 1977. There are also a number of others who are in process at one stage or another. Letters will be written again to those who are delinquent informing them their membership is in jeopardy if they do not report.

C. **CME Attendance Records**

Dr. Hastings stated that there was a problem developing in Tucson with the hospitals keeping CME attendance records for the Doctors. Discussion indicated some variance in opinion on the subject.

IT WAS MOVED AND CARRIED THAT AT THE NEXT MEETING OF THE COMMITTEE THERE BE DISCUSSION REGARDING A REQUIREMENT THAT HOSPITALS KEEP ATTENDANCE RECORDS OF PHYSICIANS AT CME PROGRAMS, AND A POSSIBLE POSITION OF ArMA CONCERNING SUCH A REQUIREMENT.

Section on Requirements

Dr. Wagner reported that the Section on Requirements had reviewed materials used by the Florida Medical association for providers of CME who wish co-sponsorship of their program by the Association. The Section feels that ArMA should develop a similar type of application for co-sponsorship, to replace our present "Request for Approval of a Program".

IT WAS MOVED AND CARRIED TO ACCEPT THE CONCEPT OF CO-SPONSORSHIP WITH NON-ACCREDITED INSTITUTIONS AND ORGANIZATIONS, IN A CAREFUL WAY IN ORDER TO ENSURE QUALITY OF CME PROGRAMS, WITH SPECIFIC PROCEDURES TO BE DEVELOPED BY THE SECTION ON REQUIREMENTS AND REPORTED TO THE NEXT MEETING OF THE MEDICAL EDUCATION COMMITTEE.

OTHER BUSINESS

Letter of 2/6/78 from Anthony Vutoro, M.D. to Louis C. Kossuth, M.D. was presented for discussion, since portions of the letter dealt with matters within the purview of this committee. Subject was a possible liaison between ArMA and the U of A College of Medicine to provide CME to the twelve outlying counties, with ArMA providing financial support for transportation, per diem and administrative personnel, the University providing faculty.

Discussion revealed some question as to the value of such programs to some outlying areas. It was also pointed out that ArMA is fiscally not in a position presently to assume such a financial responsibility. No action was taken. Dr. Kossuth will be advised of the tenor of the discussion.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc., held at 810 W. Bethany Home Road, Phoenix, Arizona on Saturday, February 25, 1978, a quorum being present, convened at 8:01 a.m., John F. Kahle, M.D., President and Chairman, presiding.

PERCENTAGE LEASES

Mr. Jacobson reviewed the 14-page legal opinion regarding the liability of Percentage Leases for Hospital-Based Physicians. It was determined to recommend to the Board of Directors that the opinion be available to the entire membership.

NATIONAL ADVISORY COUNCIL FOR THE NATIONAL HEALTH SERVICE CORPS

Discussion ensued on a possible nomination to the subject organization - No action.

JCAH

Correspondence was received seeking recommendations for the position of Director of the Hospital Accreditation Program of the JCAH - No action.

DIVIDED THEY WERE CONQUERED

Mr. RObinson reported that the estimated cost of distributing the subject report on the British National Health Service to all Arizona physicians would be approximately $1,900. Discussion ensued on the possibility of printing it in *Arizona Medicine*. It was determined to use the least expensive mechanism but to try to get the report published prior to April Ist.

STATE STEERING COMMITTEE TO IMPLEMENT THE NATIONAL VOLUNTARY COST CONTAINMENT PROGRAM

IT WAS MOVED AND CARRIED TO RECOMMEND THE FOLLOWING AS POSSIBLE APPOINTEES:

Richard L. Dexter, M.D.
Robert E. Hastings, Jr., M.D.
John F. Kahle, M.D.
Wallace A. Reed, M.D.

BRITISH NATIONAL HEALTH SERVICE SEMINAR

The invitation to send an Arizona physician to the subject seminar in London on May 12-17, 1978 was received for information.

COMMITTEE APPOINTMENTS

The question of having students and residents on the Underwriting and Claim Review Committees was discussed. It was determined that it would be best to invite students and residents to attend the Underwriting Review Committee as non-voting guests but not to invite them to the Claim Review Committee.

MEDICAL ECONOMICS COMMITTEE

Meeting of the Medical Economics Committee of the Arizona Medical Association, Inc., held Thursday, February, 16, 1978 at 810 W. Bethany Home Road, Phoenix, Arizona, convened at 7:15 p.m., Richard W. Switzer, M.D., Chairman, presiding.

ARIZONA PHYSICIAN'S GUILD

Dr. Switzer reported that the Board of Directors during meeting held December 17, 1977 reviewed the Committee's request that the Board cause to have introduced to the 1978 House of Delegates, resolution entitled "The Economic Organization for Arizona Physicians - Arizona Physician's Guild". After considerable discussion, it was determined by the Board not to introduce the resolution, but to refer it back to the Committee advising them that the Committee could introduce the resolution should it so choose.

IT WAS MOVED AND CARRIED TO INTRODUCE THE FOLLOWING RESOLUTION TO THE HOUSE OF DELEGATES FOR CONSIDERATION:

WHEREAS, The Physicians of Arizona need an effective economic organization capable of bargaining for all levels of physicians who so desire and

WHEREAS, The Arizona Medical Association as constituted can not be an effective economic organization, does not promise to be in the future and can not legally do collective bargaining in its present form and

WHEREAS, Many physicians whether salaried or self-employed want someone to represent them now and

WHEREAS, The self-employed physicians of this state need resolution of economic problems such as; many insurers not performing to contract and Federal and State regulatory bodies determining medical practice economics, therefore be it

RESOLVED: That the Arizona Medical Association in concert with the Professional Guild of Arizona create an independent collective bargaining organization and be it further

RESOLVED: That the present members of the Medical Economics Committee of the Arizona Medical Association be invited to become the temporary Executive Committee for the new organization until a general election can be held and be it further

RESOLVED: That the Arizona Medical Association will foster the growth of the new organization by referring economic problems to it (including negotiations with the Industrial Commission of Arizona) and encouraging membership.

ICA FEE NEGOTIATIONS

Dr. Switzer reported that in response to an inquiry of the Arizona Specialty Societies concerning ICA fees and use of the 1974 CRVS instead of the 1964 model, ten responses were received. The general sentiment was to increase the current ICA fee schedule considerably and to utilize the more up to date 1974 CRVS.

Mr. Beckman presented the following thoughts to be included in our presentation to the Industrial Commission this year:

A projection of the rate of inflation from external sources (President Carter's Economic Message, economists, banks, etc.) would average about 6% for 1978 and might be better received than our own estimate.

We could argue that the actual increase in the CPI was about 6.7% in 1977 which exceeded the 4.97% estimated increase in the CPI as prepared by ArMA. Therefore, an additional 1.7% could be requested to adjust for the error in last year's projection.

Federal income taxes take an increasing percentage of income as inflation pushes taxable income higher. The following exhibit illustrates how an increase in income of 7½% is needed to maintain the standard of living if living costs increase 5% for an individual with a $50,000 income. This tax situation leads to an argument that medical fees should increase faster than inflation if the doctor is to maintain his standard of living.

We might discuss the forced subsidy of one group of patients by another group if the fee schedule is inadequate. Doctors' financial requirements are fixed at any given point in time and if the revenue from industrial commission cases is inadequate, then the fees charged other patients will be raised to meet the financial requirement and industrial commission cases will be subsidized.

Another point that might be raised but might have disadvantages is that low fees may lead to restricted availability.

Also, we should point out that the practice of medicine is improving in both quality and availability. This means that previously untreatable conditions are being handled, although perhaps at high financial cost, and that people in remote areas are receiving medical care. Both of these items tend to increase the medical costs per capita without reflecting any increase in fees.

Finally, we should point out that as the average age increases, medical costs also increase because of higher utilization and not because of an increase in fees.

Considerable discussion ensued.

IT WAS MOVED AND CARRIED TO REQUEST AN 8% INCREASE IN ALL CATEGORIES OF THE INDUSTRIAL COMMISSION'S FEE SCHEDULE. FURTHER THAT THE INCREASE BE COMPUTED IN BOTH THE 1964 CRVS AND THE 1974 CRVS SUGGESTING THAT THE COMMISSION SHOULD UP-DATE ITS SCHEDULE TO THE MORE COMPREHENSIVE 1974 CRVS.

NEXT MEETING

It was determined that the committee would meet again prior to middle of April when it is anticipated the ICA will consider a new fee schedule for the purpose of reviewing the presentation.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc., held at 810 W. Bethany Home Road, Phoenix, Arizona on Friday, March 17, 1978, a quorum being present, convened at 6:45 p.m., John F. Kahle, M.D., President and Chairman, presiding.

ATTORNEY GENERAL DEMANDS

Mr. Daniel J. McAuliffe, Snell & Wilmer, legal counsel, reviewed the recently served State of Arizona Attorney General "Demands for Production of Documents or Tangible Objects" and "Demand To Answer Written Interrogatories."

He explained that he had negotiated a number of the items with the Attorney General's office so that the demands were less cumbersome and less lengthy than originally presented. Discussion of fighting in court the demands to the Association ensued, but it was agreed that success was limited and could be counter productive at this time.

Mr. Robinson indicated that it will take several weeks of his full time to respond to the demands as well as thousands of copies of documents.

COMMITTEE STRUCTURE

The Committee reviewed the Finance Committee's recommendations that all committee structures be reviewed for size and effectiveness.

IT WAS MOVED AND CARRIED TO THANK THE FINANCE COMMITTEE AND ADVISE THEM THAT THIS IS BEING DONE ON AN ANNUAL BASIS BY THE PRESIDENT.

PHYSICIAN PLACEMENT SERVICE

The Finance Committee's recommendation that we charge for the placement service was discussed at some length.

IT WAS MOVED AND CARRIED TO PROVIDE THE FIRST REQUESTED LISTING OF OPPORTUNITIES FREE BUT TO CHARGE A $10 FEE FOR ALL SUBSEQUENT LISTINGS ISSUED IN A ONE-YEAR PERIOD. THIS POLICY IS NOT TO BE IMPLEMENTED UNTIL IT IS CLEARED AND APPROVED BY THE AMA WHICH PROVIDES US WITH THE BASIC DATA.

NATIONAL COMMISSION ON THE COST OF MEDICAL CARE

The question of whether the Summary Report from the subject group should go to ArMA's House of Delegates in April was discussed.

It was determined that Dr. Kahle would contact Dr. Hastings, Chairman of our Ad Hoc Committee on Health Care Cost, and ask him to have his committee review the report with the intent of making recommendations in resolution form to be presented to the House of Delegates in April.

ARIZONA HEALTH SERVICE CORPS

Mr. Robinson reported on the activities relating to recovering money paid to Ward B. Studt, M.D. which was subsequetly denied by H.E.W., summarized as follows:

A. Executive Committee action of 1/20/78 "Exhaust all avenues of collecting these funds from HEW before going to Dr. Studt for reimbursement. A time frame of three months was set for this activity"

B. Letter to Civil Service Commission 1/6/78

C. Letter to Dr. Studt 1/30/78 asking for his help and advising him of the Executive Committee action of 1/20/78

D. Follow-up letter to Civil Service Commission 2/23/78

E. Letter to Dr. Nichols seeking his help 3/10/78

F. Phone call to Dr. Nichols to discuss the matter during which he said that he would try to help.

It was recommended that additional calls be made to Mr. Fedorowick and Dr. Nichols trying to resolve the problem.

It was also determined that a follow-up letter, certified with a return receipt requested, be sent to Dr. Studt to see if he has had any success in resolving this problem.

COMPENSATION REVIEW

IT WAS MOVED AND CARRIED TO APPROVE A 7½ PERCENT SALARY INCREASE FOR THE EXECUTIVE DIRECTOR EFFECTIVE APRIL I, 1978.

CANADIAN HEALTH SERVICE STUDY

Dr. Kahle reviewed the request from James H. Sammons, M.D., Executive Vice President of the AMA that Bruce E. Robinson be permitted to participate in a study of the Canadian Health Service April 17-21, 1978.

IT WAS MOVED AND CARRIED TO APPROVE THE REQUEST THAT BRUCE E. ROBINSON BE ABLE TO PARTICIPATE IN THE STUDY OF THE CANADIAN HEALTH SERVICE.

MATERNAL & CHILD HEALTH CARE COMMITTEE

The meeting of the Maternal & Child Health Care Committee of the Arizona Medical Association, Inc., held at 810 W. Bethany Home Road, Phoenix, Arizona on Saturday, March 18, 1978, convened at 1:43 p.m., William C. Scott, M.D., Chairman, presiding.

BOARD OF DIRECTORS

The Chairman reviewed the actions of the Board of Directors on recommendations which emanated from this committee's meeting of December 10, 1977.

PLANNED PARENTHOOD

The committee discussed at greath length the request from Planned Parenthood that we endorse a document entitled "Planned Births, the Future of the Family and the Quality of American Life."

IT WAS MOVED AND CARRIED TO REFER THIS DOCUMENT TO ALL COUNTY MEDICAL SOCIETIES IN ARIZONA AND THE ARIZONA OSTEOPATHIC MEDICAL ASSOCIATION ASKING FOR THEIR COMMENTS AND RECOMMENDATIONS.

LAY MIDWIFERY RULES — LEGAL OPINION

The Chairman reviewed the requested legal opinion on the liability physicians may incur should they participate with lay midwives under the D.H.S. rules adopted December 5, 1977. Extensive discussion ensued over the entire subject of lay midwives and the implications they have on good medical care.

IT WAS MOVED AND CARRIED TO DISSEMINATE THE OPINION TO THE ENTIRE MEMBERSHIP.

The Chairman indicated that he would draft the material to be placed in *Medical Memos.* It was also agreed that MICA should be given a copy of the regulations and opinion.

AMA'S MATERNAL & CHILD HEALTH CARE COMMITTEE MEETING

Dr. Scott extended an invitation to the committee to attend the subject meeting scheduled for April 29-30, 1978 at the Hyatt Regency in Phoenix.

Meeting adjourned 3:04 p.m.

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education.

Arizona Heart Institute
Arizona Medical Association, Inc.
Arizona State Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates
Phoenix Indian Medical Center
Phoenix Psychiatric Council
Maricopa County General Hospital, Phoenix
Memorial Hospital, Phoenix
St. Luke's Hospital & Medical Center, Phoenix
St. Joseph's Hospital & Medical Center, Phoenix
Tucson Hospitals Medical Education Program, Tucson
U of A College of Medicine
Veterans Administration Center, Prescott
Veterans Administration Hospital, Phoenix

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

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Individual program listings below indicate Category of CME. Where not indicated, no credit has been approved. For further information on any programs, contact person named.

JUNE

17th Annual State Convention
Maricopa Chapter, American Association of Medical Assistants, June 9, 10, 11, 1978, Doubletree Inn, Scottsdale, AZ. Sponsor: Maricopa Chapter, American Association of Medical Assistants. Contact: Jan Neal, CMA Convention Chairman, 4502 N. 36th St., #108, Phoenix, AZ 85018.

A Day of Surgery
July 28, 1978. Little America Hotel, Flagstaff, AZ. Sponsor: College of Medicine, AZ Chapter of American College of Surgeons. Contact: C.H. Fredell, M.D., 120 W. Fine St., Flagstaff, AZ 86001. Approved for 7 hours of category 1 credit.

General Staff Quarterly Meeting
June 21, 1978. Chimney's Restaurant, Glendale, AZ. Sponsor: Glendale Samaritan Hospital. Contact: Richard Jaynes, M.D., Glendale Samaritan Hospital, 6010 West Northern, Glendale, AZ 85301. Approved for 1 hour of category 1 credit.

Medical Horizons: Advances in Therapeutics
June 30-July 2, 1978, Little America Hotel, Flagstaff, AZ. Sponsor: College of Medicine, U. of A. Contact: Anthony F. Vuturo, M.D., Office of Continuing Medical Education, U of A, Tucson, AZ 85724. Approved for 10 hours of category 1 credit.

JULY

21st Annual Ruidoso Family Practice Seminar
July 17-20, 1978. Headquarters Inn, of the Mountain Gods at Mescalero, New Mexico. Sponsors: New Mexico Chapter of the American Academy of Family Physicians. Contact: Bob Reid, Convention Dir., P.O. Box 456, Sunland Park, N.M. 88063. Aapproved for category 1 credit.

First Annual Summer Pediatric Conference Preparation for Recertification
July 7, 8, 9, 1978. 7:30 a.m.-2 p.m., Little America, Flagstaff, AZ. Sponsor: CME, U of A Health Sciences Center. Contact: Anthony F. Vuturo, M.D., ASHSC, 1501 N. Cmapbell Ave., Tucson, AZ 85724. Approved for 17.5 hours category 1 credit.

Coconino County Medical Society 14th Summer Medical Semlnar
July 29 & 30, 1978. Little America, Flagstaff, AZ. Sponsor: Coconino County Medical Society. Contact: Paul Lundstrom, M.D., 120 W. Fine, Flagstaff, AZ 86001. Approved for 8 hours of category 1 credit.

MONTHLY OR WEEKLY

Headache Rounds
Tuesday afternoons or Thursday mornings weekly. Sponsor: Headache Group of the Southwest. Contact: Peggy Voigt, 2610 W. Bethany Home Rd., Phoenix, AZ 85017, or phone 242-6646.

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m., J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8-10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., Arizona Medical Center, Dept. of Radiology, Tucson AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m. in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology, Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

Staff Education Conference
Wednesdays, weekly, 1 p.m., Arizona State Hospital, Phoenix, AZ. Sponsor: Arizona State Hospital. Contact: Howard E. Wulsin, M.D., Arizona State Hospital, 2500 E. Van Buren, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Road, Phoenix, AZ 85013. Sponsor: St. Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

Neurosurgery Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.

Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Room—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists.

Neuropathology of Gross Specimens Conference
Thursdays, 7:30-8:30 a.m. in the Morgue.

Neurology-Neurosurgical Clinical-Neuroradiological and Neuropathological Conference
Fridays, 8-9 a.m., First Floor Conference Room.

Neuropathology or Neuroradiology Conferences
Fridays, 9 a.m., Neuropathology in Neuropathology Conference Room, Neuroradiology in First Floor Conference Room.

Neurosurgical Journal Club
Saturdays, 9-11 a.m., in Eighth Floor Conference Room.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.

Patient Staffing Conference
Three times weekly.

GOOD SAMARITAN HOSPITALS

Desert Samaritan Hospital
Wednesday evenings, 7 p.m. Sponsor: Desert Samaritan Hospital. Contact: L.A. Rosati, M.D., Laboratory, Desert Samaritan Hospital, Mesa, AZ 85202. Approved for category 1 credit.

Clinical Cancer Conference
3rd Wednesday, every month, Butler Bldg. Conference Room, Good Samaritan Hospital. Contact: John A. Bruner, M.D., 926 E. McDowell Road, Phoenix, AZ 85006. Approved for 1 hour per session category 1 credit.

Pulmonary Disease Grand Rounds
Mondays, 12 Noon, D-5 North Conference Room, Good Samaritan Hospital, Phoenix, AZ. Sponsor: Pulmonary Disease Teaching Service, Good Samaritan Hospital. Contact: Bernard E. Levine, M.D., Pulmonary Function Laboratory, Good Samaritan Hospital, 1033 E. McDowell Road, Phoenix, AZ 85006. Approved for 1 hour category 1 credit.

Continuing Medical Education Program
Maryvale Samaritan Hospital, every other Wednesday, 7-8 a.m., January 25, 1978 through December 27, 1978. Sponsor: Medical Staff of Maryvale Hospital. Contact: Richard C. Christensen, M.D. Approved for 1 hour per session category 1 credit.

MEMORIAL HOSPITAL

1201 S. 7th Ave., Phoenix, AZ 85036. Sponsor: Memorial Hospital. Approved for category 1 credit.

Monthly Medical Education Seminar
Third Monday, 6:30 p.m., Kiva Conference Room. Contact: George Scharf, M.D.

Tumor Board Conference
Every Friday at Noon, Kiva Conference Room. Contact: Howard Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 E. 4th St., Scottsdale, AZ 85251. Sponsor: Scottsdale Memorial Hospital. Contact: R.C. Good, M.D., Director of Medical Education.

Clinical Pathological Conference
4th Monday, monthly, 12:45 p.m.

Family Practice Conference
1st Monday, monthly, 12:45 p.m.

Medical Grand Rounds
3rd Monday, monthly, 12:45 p.m.

Morbidity & Mortality Conference
2nd Monday, monthly, 12:45 p.m.

ST. MARY'S HOSPITAL & HEALTH CENTER

1601 West St. Mary's Rd., Tucson, AZ 85703. Sponsor: St. Mary's Hospital & Health Center. Contact: see below.

Monthly Specialty Conference of the Department of Surgery
First Monday, monthly, 7:30 a.m., Century Room A. Contact: Medical Staff Office. Approved for 1 hour of category 1 credit.

Grand Rounds: Medical, Surgical, Family Practice, Pathology, Radiology
Weekly, Thursday, 7-8:30 a.m. Contact: Richard A. Silver, M.D. Approved for 1 hour category 1 credit.

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Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. A brief summary follows:

* **Warning**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome, Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

Supplied: Bottles of 100 and 1000 capsules; Single Unit Packages of 100 (intended for institutional use only).

FOR LONG-TERM CONTROL OF HYPERTENSION.* SERUM K^+ AND BUN SHOULD BE CHECKED PERIODICALLY. (SEE WARNINGS SECTION.)

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U OF A HEALTH SCIENCES CENTER

Tucson, AZ 85724. Sponsor: U of A College of Medicine. Contact: See below. Approved for category 1 credit.

Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 p.m. Contact: John Boyer, M.D., Dept. of Internal Medicine.

Cerebrovascular Disease Conference
Mondays, 5-6 p.m., weekly, Room 5505. Contact: Wesley Moore, M.D., Depts. of Surgery, Neurology & Radiology.

Dean's Clinical Rounds
July 19, 1978, Main Auditorium, UAHSC. Contact: Louis J. Kettel, M.D.

Endocrinology Seminar
Every Thursday, Noon-1 p.m., 1st, 3rd & 5th Thursday, Room N318, VA Hospital. 2nd & 4th Thursday, Room 6505, Tucson Medical Center. Contact: Dept. of Internal Medicine.

Hematology-Oncology Clinical Conference
Every Tuesday, Noon-1 p.m., 1st, 3rd & 5th Tuesdays, Room 6505, Tucson Medical Center. 2nd & 4th Tuesdays, Room N318, VA Hospital. Contact: Sidney Salmon, M.D., Dept. of Internal Medicine.

Medical Grand Rounds
Every Wed., Noon-1 p.m., 1st, 3rd & 5th Wed., Staff Conf. Room, VA Hospital. 2nd & 4th Wed., Room 5403, UAHSC. Contact: Jay Smith, M.D., Dept of Internal Medicine.

Nuclear Medicine
Thursday, 7:30 a.m., Radiology Conf. Room, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept of Diagnostic Radiology.

Neurology Grand Rounds
Every Friday, Noon, 1st, 3rd & 5th Fridays, UAHSC, Room 7505. 2nd & 4th Fridays, VA Hospital. Contact: William A. Sibley, M.D., Dept. of Neurology.

Neuropathology Review Conference
Thursdays, 8:30 a.m., Room 5120, UAHSC. Contact: Peter C. Johnson, M.D.

Neurosciences Seminar
Every Friday, 7:30 a.m., UAHSC, Room 7505. Contact: Colin R. Bamford, M.D., or Jose F. Laguna, M.D., Dept. of Neurology.

Pathology Seminar
Friday, 4:30-5:30 p.m., Room 5120, UAHSC. Contact: Kenneth Ryan, M.D., Dept. of Pathology.

Tucson Pathologist Conference
First Monday of the month, 7:30-9 p.m. Rotation between Tucson Pathology Conf Rooms. Contact: Merel Clard, M.D., Tucson Pathologists.

Pediatric Grand Rounds
Every Tuesday, 12:30-1:30 p.m. Alternates between Tucson Medical Center & UAHSC. Contact: Hugh Thompson, M.D., Dept. of Pediatrics.

Problem Case Workshop
3rd Monday of each month, 7:30 a.m., Room 4410, UAHSC. Contact: H.E. Cross, M.D., Ph.D., Div. of Ophthalmology.

Psychiatric Grand Rounds
Every Wednesday, Sept. to May 4, 5:30 p.m., Room 8403 UAHSC. Contact: Alan Levenson, M.D., Dept. of Psychiatry.

Chest Radiology
Monday, 5-6 p.m., weekly, Room 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Gastrointestinal Radiology
Mondays, 7:30 a.m., weekly except for summer months. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Radiation Oncology Planning Conference
Friday, weekly, 8:30-10 a.m., UAHSC, Room 0655. Contact: Robert S. Heusinkveld, M.D., Radiation Oncology Section.

Radiology-Rheumatology Conference
Thursday, 8 a.m., UAHSC, Library Room 1535C. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Vascular Radiology
January-June 1978, 7:30 -8:30 a.m., every Friday. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Radiology Interesting Case Conference
Thursday, Noon, VA Hospital. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Room. Contact: Irwin M. Freundlich, M.D., Dept of Radiology.

Trauma Conference
Every Thursday, 4-5 p.m. UAHSC, Room 5505. Contact: Martin Silverstein, M.D., Dept. of Surgery.

Grand Ward Rounds—Trauma
Every Tuesday, 8 a.m., UAHSC. Contact: Martin Silverstein, Surgery Dept., Trauma Section.

Monthly Meeting Tucson Ultrasonography Group
First Wednesday of month, 4:30-5:30 p.m., UAHSC. Contact: Irwin M. Freundlich, M.D.

Tucson Urological Society Scientific Meeting
Monthly. Contact: George W. Drach, M.D., Depts of Surgery & Urology.

General Urology Conference
Tuesday, 5-6 p.m. Thursday, Noon-1:30 p.m., UAHSC Meeting Rooms & VA Hospital. Contact: George W. Drach, M.D., Dept. of Surgery/Urology Section.

VETERANS ADMINISTRATION HOSPITAL

7th Street and Indian School Rd., Phoenix, AZ 85012. Sponsor: VA Hospital. Contact: Jasper L. McPhail, M.D. Approved for category 1 credit.

Cancer Symposium
Each Wednesday, 1 p.m., T-5.

Cardiology Conference
2nd Thursday, 1 p.m., T-5.

Medical-Surgical Chest Conference
1st and 3rd Thursday, 1 p.m., T-5.

Clinicopathology Conference
4th Thursday of 3rd Mo. (Mar., June, Sept. & Dec.), 1 p.m., T-5.

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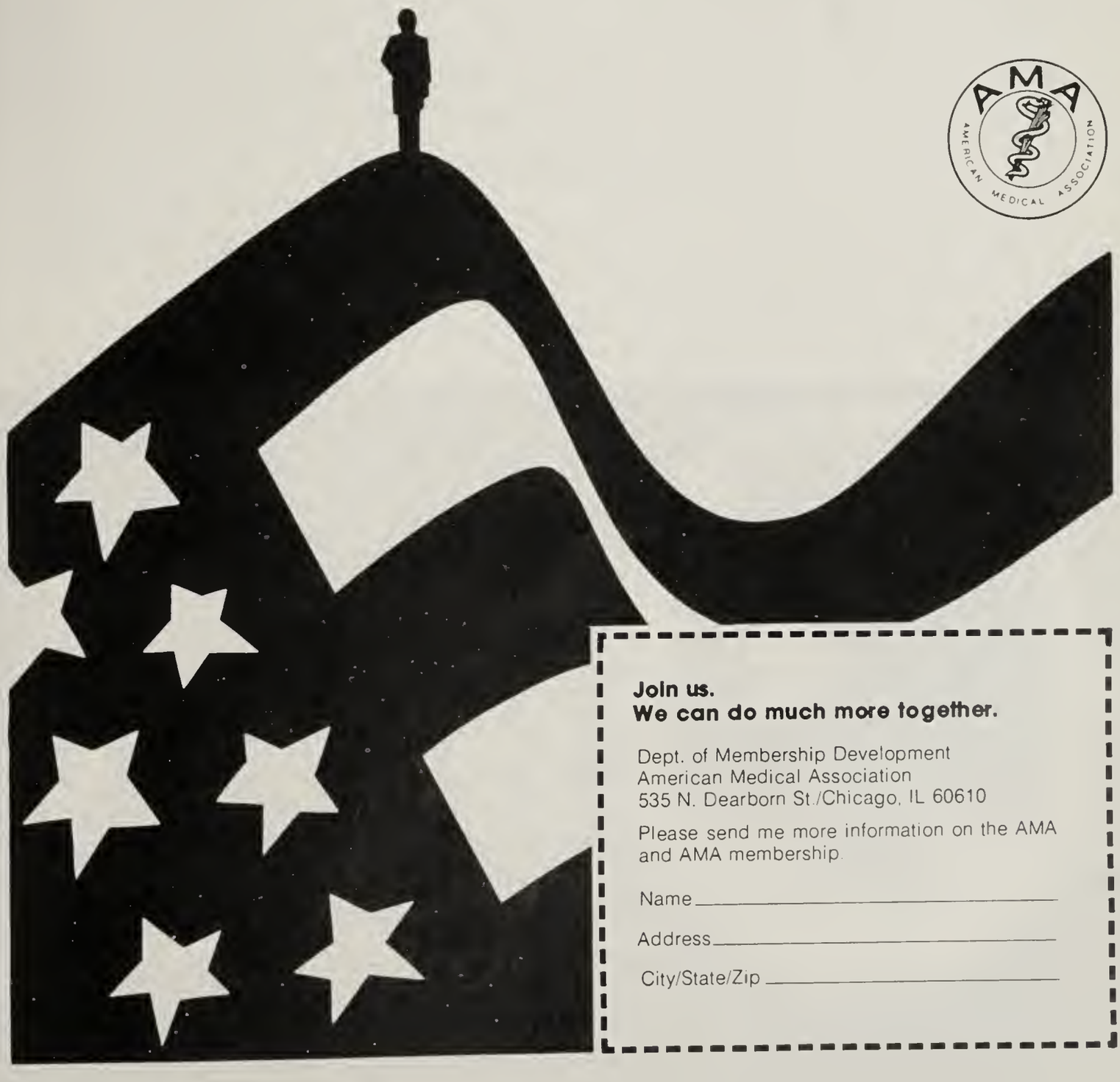
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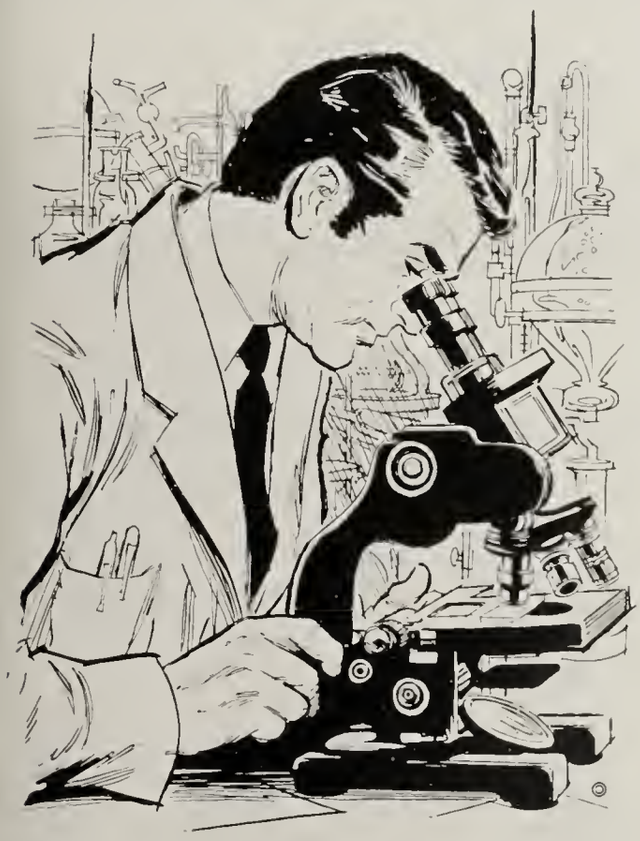
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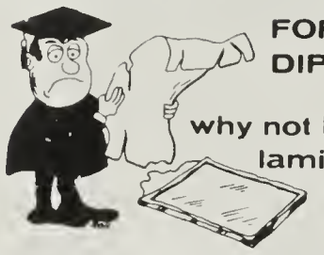
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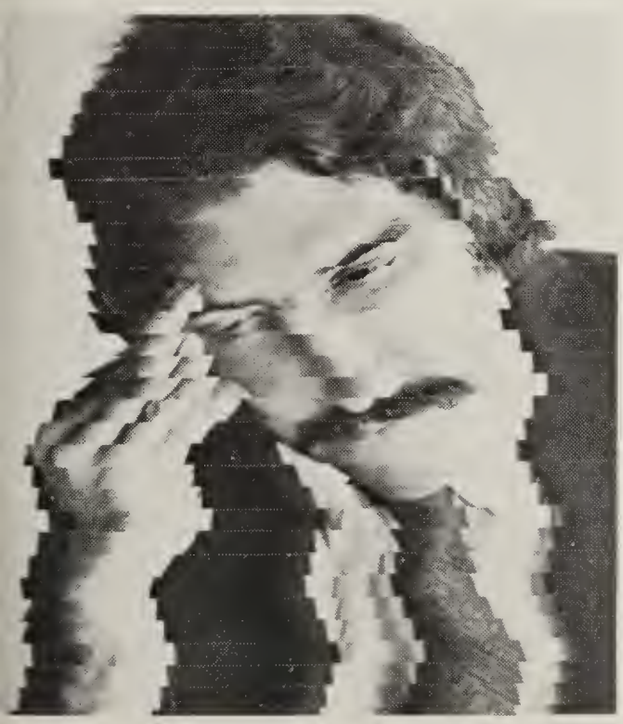
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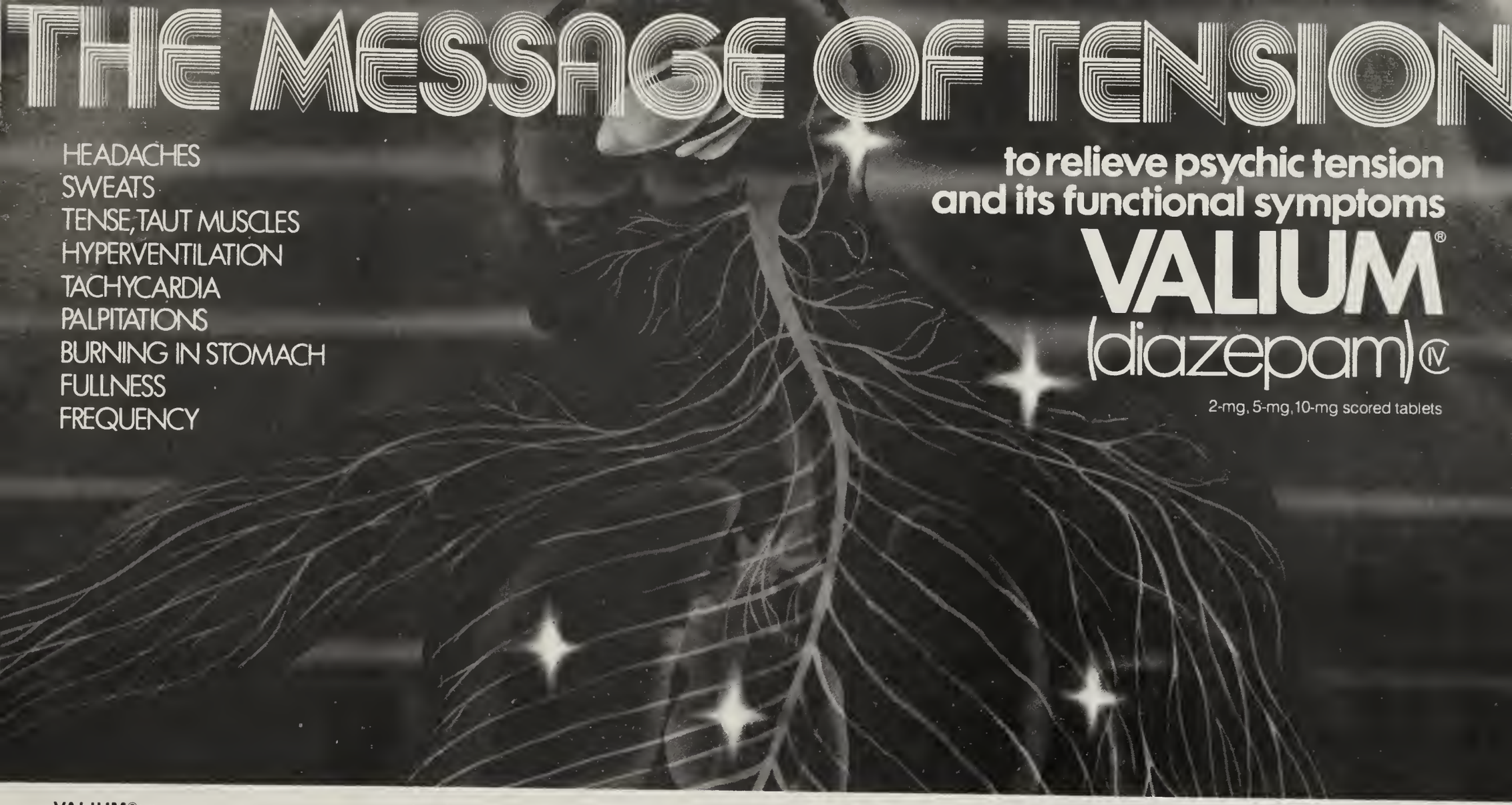
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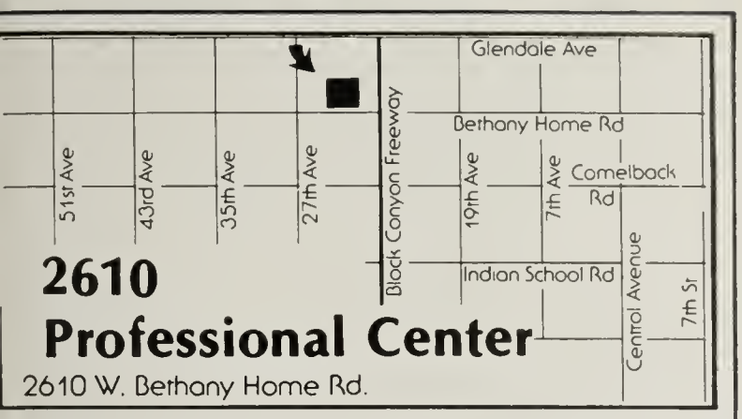
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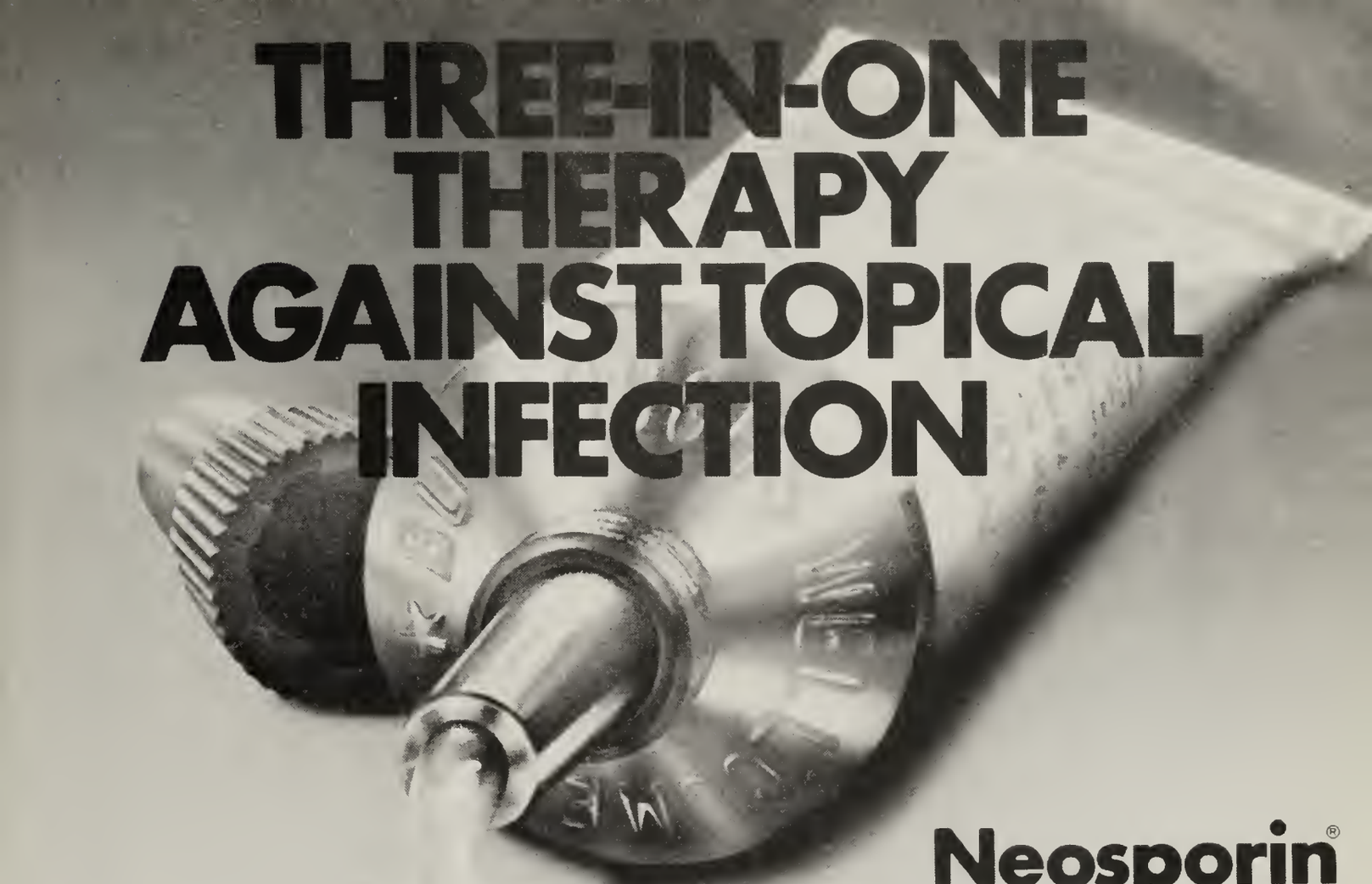
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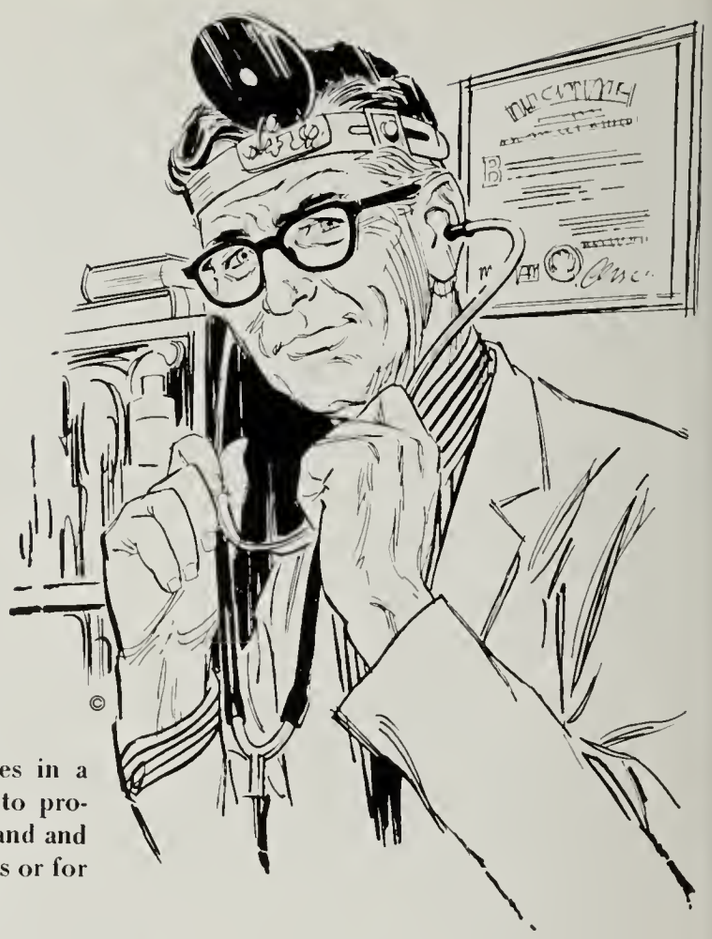
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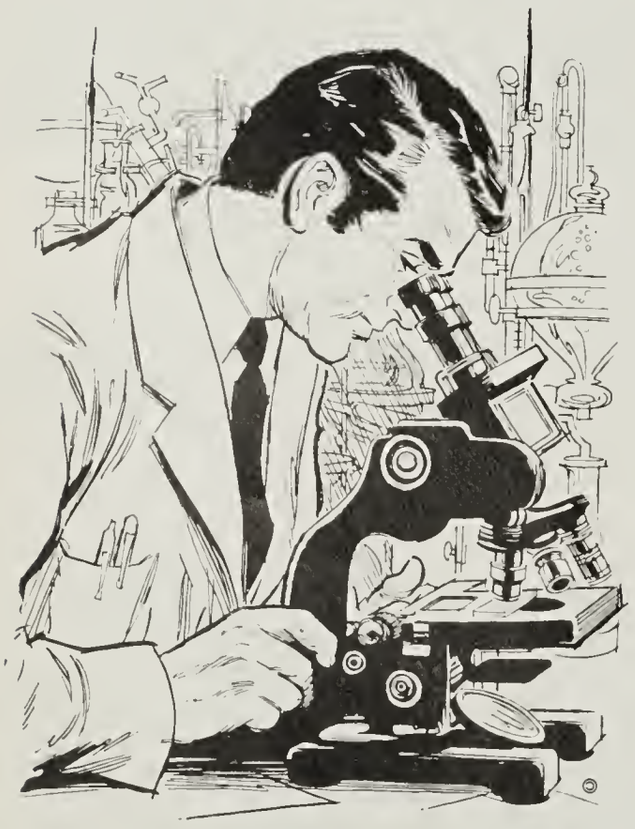
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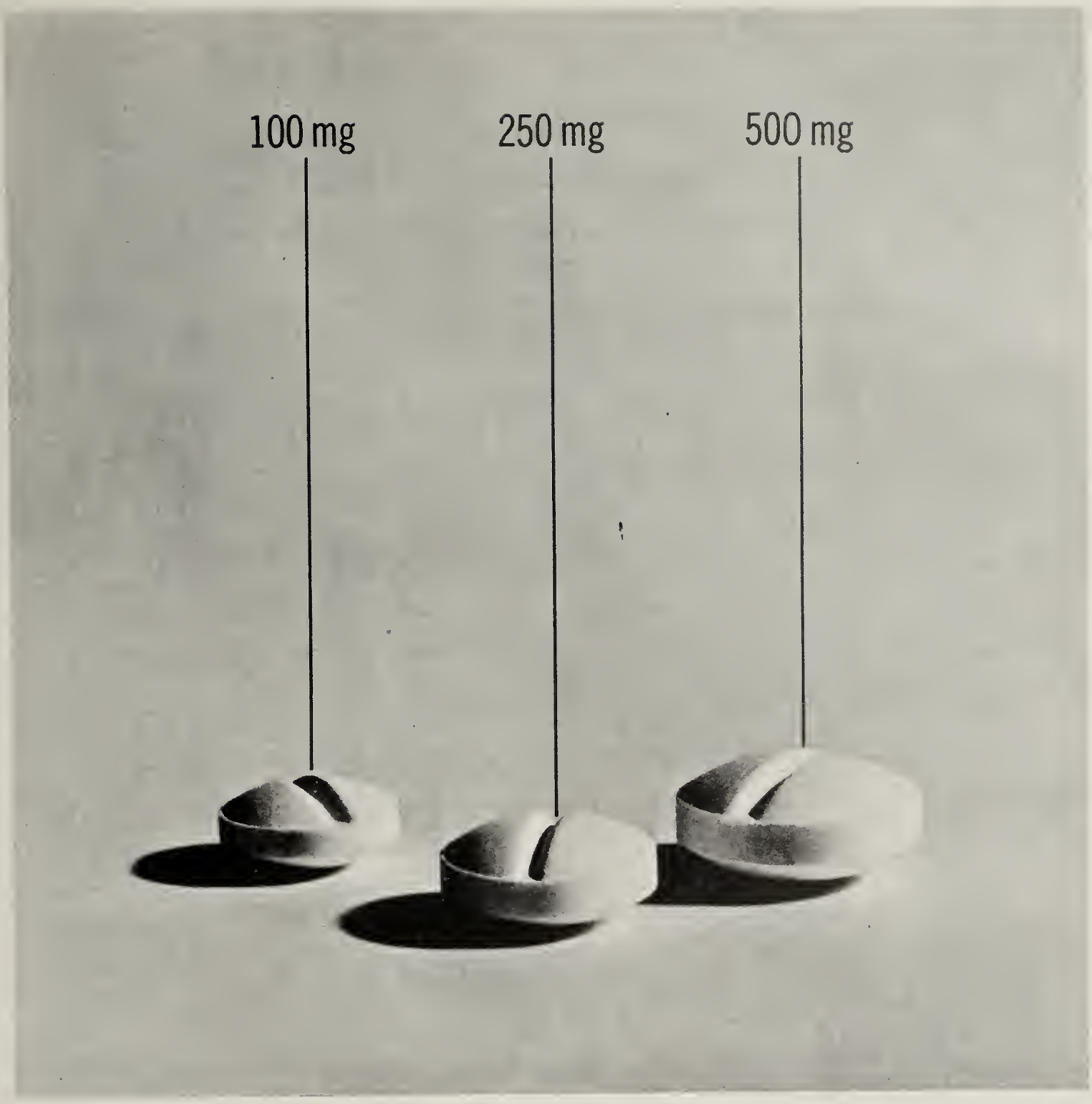
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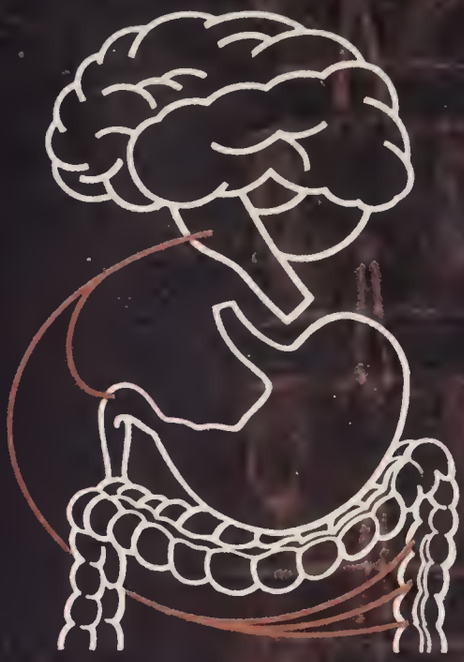
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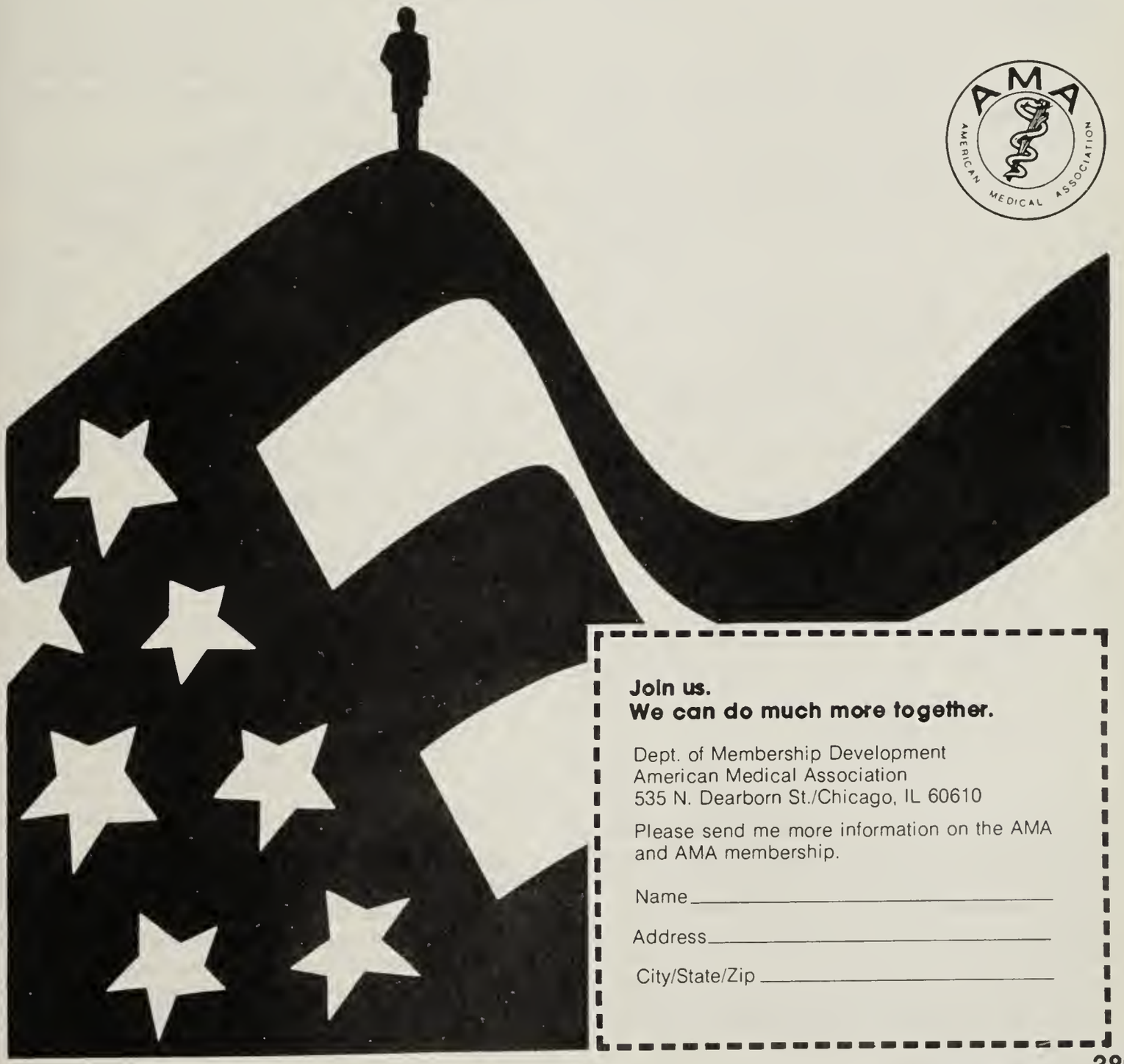
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Coronary Artery Surgery:

For related articles see page 414.

Lee B. Brown, M.D.
Alberto Benchimol, M.D.

INTRODUCTION

The prevalence of coronary artery disease is well-known. Disability and death resulting from coronary artery disease are enormous.[4,16,6]

Aortocoronary bypass has become a commonplace operation; and it has been estimated that in excess of 50,000 operations are performed in the United States on a yearly basis.[26]

The purpose of this paper is to enter a status report on coronary artery surgery that we have been involved with at Phoenix's Good Samaritan Hospital.

RESULTS OF STUDY

One hundred consecutive aortocoronary bypass procedures performed between April 7, 1975 and November 3, 1976 were selected for study.

TABLE I — Cases Excluded.
Simultaneous Valve Replacement
5 cases - 2 deaths

AVR* + 1 bypass	1
AVR + 2 bypasses	1
MVR* + 1 bypass	1
MVR + 2 bypasses	1
AVR + MVR + 1 bypass	1
	5

*AVR = Aortic Valve Replacement
*MVR = Mitral Valve Replacement

Five cases which resulted in two deaths were excluded. Reference should be made to Table 1. In each instance, aortocoronary bypass was combined with either single or multiple valve replacement. The primary purpose of surgery in each instance was valve replacement.

With respect to the 100 consecutive aortocoronary bypass operations performed between April 7, 1975 and November 3, 1976, there was one death. The operative mortality is 1%. If cases in which simultaneous valve replacement are included, there were 3 deaths. The operative mortality rate is 3%.

There have been no late deaths.

TABLE II - Age

Average age	55 years
Age range	27-70 years
Age groups:	
20-29	1
30-39	6
40-49	20
50-59	42
60-69	28
70+	3
	100

TABLE III — Sex

Male	88
Female	12
	100

TABLE IV - Classification Angina

Stable	0
Unstable	100
Class I	- 66
Class II	- 25
Class III	- 9

All subsequent data given refers to the subject of this report - the 100 consecutive aortocoronary bypass operations between April 7, 1975 and November 3, 1976.

Table II gives various information related to age. The average age was 55 years with an age range from 27 to 70.

Table III indicates there were 88 males and 12 females.

Table IV details the clinical classification of angina. Angina can be classified as stable or unstable. Unstable

From: Good Samaritan Hospital, Phoenix, AZ. Reprint requests to Dr. Brown, 926 E. McDowell Rd., Phoenix, AZ 85006.

angina refers to a changing pattern of angina in either severity or frequency, or both.

Unstable angina is customarily referred to by a variety of terms which denote the severity of the syndrome. We have divided angina into Class I, II and III. Class I refers to patients with definite but modest changes in the pattern of angina, or the recent onset of angina. Class II refers to patients with a more abrupt and more severe change in the pattern of angina. Crescendo angina and accelerated angina are synonymous terms. Class III refers to patients with a still more severe and rapid progression of angina. Pre-infarction angina is a synonymous term. The clinical issue in patients with Class III angina is whether or not myocardial infarction has occurred.

TABLE V - Associated Conditions

Previous MI*	44
Previous CHF**	5
Hypertension	8
Aortocranial occlusive disease	4
Abdominal aneurysm	2
Peripheral vascular disease	4
Hyperlipidemia	2
Diabetes	6
Emphysema	4
Other	2

*MI - Myocardial Infarction
**CHF - Congestive Heart Failure

TABLE VI - Preoperative ECG and VCG

Previous MI	44
Ischemia	29
VPC, frequent	4
1° AV block	3
2° AV block	2
Normal or "borderline"	25

When coronary arteriography and consideration of surgical treatment are judged advisable, Class I patients are managed in an elective fashion, Class II patients as promptly as possible, and Class III patients are generally admitted directly and urgently to the coronary unit.

Table V lists relevant associated conditions.

Table VI categorizes the interpretation of the preoperative electrocardiogram and vectorcardiogram. Forty-four patients had changes indicative of a previous myocardial infarction. Ischemia, without definite evidence of myocardial infarction, was the interpretation in 29 patients. Frequent and persistent VPC was noted in 4 patients. A normal or "borderline" interpretation was recorded in 25 patients.

TABLE VII - Cardiac Catheterization: Vessel Disease

Single	9
Double	27
Triple	37
Triple "plus"	12
Double or triple + main	15
	100

In Table VII is recorded the extent of vessel involvement. Single, double, and triple refers to a single lesion of the circumflex, anterior descending or right coronary, either alone or in any combination. Reference to triple "plus" refers to involvement of both the marginal and posterior descending of the circumflex, or it refers to triple vessel disease with the addition of the diagonal branch of the anterior descending. In 15 patients, there was significant involvement of the main left coronary, 50% or greater, in addition to double or triple vessel disease. Although a significant lesion of any coronary was taken to be 70% occlusion or greater, somewhat lesser involvement of one coronary may have existed in association with multiple vessel involvement.

TABLE VIII - Cardiac Catheterization: Other Data

LVEDP	>15	27
CI	< 2	44
L.V. aneurysm		10
Dyskinetic ventricle		56
1 wall	46	
2 walls	10	

Table VIII records additional information derived from cardiac catheterization. Ejection fractions were not routinely done during the period of this study. The assessment of akinetic or dyskinetic segments of left ventricle was made from the ventriculogram in the right anterior oblique projection.

TABLE IX - Surgical Procedures

Single bypass	8
Double bypass	40
Triple bypass	44
Quadruple bypass	8
	100
252 grafts in 100 patients = 2.5 grafts/patient	
IMA used	31

The surgical procedures performed, with reference to aortocoronary bypass, are detailed in Table IX. Triple or quadruple bypass was performed in 52 patients. There were 252 grafts in 100 patients or 2.5 grafts per patient. The internal mammary artery was utilized to bypass either the anterior descending or the diagonal branch of the anterior descending in 31 patients.

TABLE X - Combined Surgical Procedures

Resection L.V. Aneurysm	8
Carotid Endarterectomy	2
Permanent Pacemaker	2
IAPB*	6
Excision Granuloma, Lung	1

*IAPB = Intraaortic balloon counterpulsation

Operative procedures combined with aortocoronary bypass are listed in Table X. Resection of left ventricular aneurysm was performed when there was parodoxical motion, when there was inverting of the aneurysm with left ventricular suction, or when there was either clinical evidence of mural thrombus, or when there was evidence of mural thrombus on the basis of the ventriculogram. Carotid endarterectomy was combined with aortocoronary bypass in 2 patients. Six patients required the use of intraortic balloon counterpulsation (IABP). IABP was

TABLE XI - Complications

Perioperative Infarction (POMI)	8
Bleeding	14
Renal failure	2
Arrythmia	16
AF* 12	
VT - VPC* 4	
Thrombophlebitis	5
Pulmonary	2
Brain injury, transient	1

*AF = Atrial fibrillation
VT = Ventricular tachycardia
VPC = Ventricular premature contraction

initiated prior to cardiopulmonary bypass in 3 patients, and in 3 additional patients IABP was implemented in the immediate period following termination of cardiopulmonary bypass.

The complications of surgery are listed in Table XI.

An aggressive attitude with respect to bleeding is evident. Fourteen patients were explored for bleeding. Although there were occasional variations, if bleeding exceeded 1500 cc's in 12 hours, 200 cc's per hour for four hours, 250 cc's an hour for two hours, or greater than 350 cc's in one hour, the reoperation was performed. Determination of partial thromboplastin time, prothrombin time, platelet count, fibrin split products, and clotting time were routinely performed when excessive bleeding was noted. Significant variations from normal were rarely noted.

Eight patients had electrocardiographic evidence of perioperative infarction (POMI) and this was confirmed by vectorcardiogram in the later postoperative course. Two of the 8 patients had left ventricular power problems. All 8 patients made a good recovery.

Dialysis was required for renal failure in 2 patients. Both made a good recovery.

Arrythmia was noted in 16 patients. Atrial fibrillation occurred in 12 patients. Frequent ventricular premature beats and/or ventricular tachycardia occurred in 4 patients. Most responded to conventional drug management; countershock was often employed for both atrial and ventricular arrythmias.

The significant incidence of thrombophlebitis has caused us to avoid cannulation of the femoral vein.

Transient mild pulmonary problems were frequent. Two patients had pulmonary problems which were salient in postoperative care, and required prolongation of the customary respiratory support measures.

One patient died in the 30 day period following operation. Patient C.R. was a diabetic male, age 52, who underwent triple aortocoronary bypass. The operative procedure was well tolerated, but reoperation for bleeding was necessary in the early postoperative period. His early recovery was quite satisfactory. Some ten days following the operation, he developed bilateral pulmonary infiltrates which progressed, and later became consolidated and cavitary. Steady clinical deterioration was associated with progressive pulmonary disease. Repeated sputum cultures failed to disclose a significant pathologic organism. Lung biopsy was performed on the 25th postoperative day and the diagnosis was Nocardia brasiliensis. Death occurred on the 34th postoperative day due to pulmonary sepsis and pulmonary failure. In the last few days of life, ventricular arrhythmias appeared which became uncontrollable in the final hours of life. Autopsy examination was performed and pneumonic lesions due to Nocardia were demonstrated; and, in addition to this, purulent Nocardia pericarditis and thrombosis of two of the three bypass grafts were present.

Nine patients had *recurrent angina* between 2 and 9 months postoperatively. Repeat cardiac catheterization revealed that *13 of 22 grafts were closed*. Surprisingly, no ECG or VCG changes were noted in 8 patients.

Two patients included in this group of 100 patients had had previous aortocoronary bypass.

Recurrent angina was noted 7 months following operation in one patient who had undergone previous triple aortocoronary bypass. Saphenous graft had been used for bypass of the posterior descending division of the circumflex, anterior descending, and right coronary. Coronary anteriograms disclosed occlusion of the saphenous graft to the anterior descending. Reoperation consisted of internal mammary to anterior descending bypass. There has been no recurrence of angina in the 11 months following reoperation.

The second patient, a physician, developed recurrent angina 5 months following saphenous graft bypass of the posterior descending division of the circumflex and the anterior descending. Both grafts were proven occluded upon repeat coronary arteriography. Reoperation consisted of saphenous vein bypass of the posterior descending of the circumflex, left internal mammary bypass of the diagonal branch of the anterior descending and right internal mammary bypass of the terminal portion of the anterior descending. There has been no recurrence of angina in the 13 months following reoperation.

Recurrent angina was noted in 1 patient with 2 of 2 grafts patent. There was a documented perioperative infarction recorded in the early postoperative period.

Five patients without symptoms underwent repeat cardiac catheterization. Nine of 9 grafts were noted to be patent.

DISCUSSION

The operative mortality of 1% is acceptably low. It is not, however, unusual. There are numerous reports which record a similar or better experience. (Table XII)[2,3,9,18,22,23,35,37]

The complications are certainly significant; but at the same time, they are usually transient. Few complications resulted in ongoing disability. The exceptions include the 10 patients with recurrent angina with or without graft occlusion and the one death that resulted from pulmonary sepsis.

Perioperative myocardial infarction (POMI) is viewed as the most important complication of aortocoronary bypass. The exact reported incidence of this complication

TABLE XII - Aortocoronary Bypass Operative Mortality (30 days)

Author	Date	Ref.	# Cases	Op. mortality
Wisoff	1975	(37)	200	0.0
Hutchinson	1975	(18)	376	0.8
Cheanvechai	1974	(9)	545	2.5
Kouchoukos	1977	(22)	1514	1.6
Ullyot	1977	(35)	200	0.5
Keon	1976	(23)	930	4.9
Assad-Morrell	1976	(2)	500	3.0
Berg	1975	(3)	1612	2.8

varies between 2% and 13%. (Table XIII)[2,3,9,18,22,28,35,57] Although we found that occlusion of at least one graft is usually noted in patients with recurrent angina, patients who sustained perioperative myocardial infarction were not systematically restudied. Fruehau et al.[14] restudied 11 of 36 patients who survived POMI. Seven of the 11 patients had all grafts patent, and it was concluded that graft occlusion is not necessarily associated with POMI. The findings of Buckley et al.[8] were similar.

A review of the recorded complications indicates the requirement placed upon the surgical team, medical team,

TABLE XIII - Perioperative Myocardial Infarction (POMI)

Author	Date	Ref.	# Cases	POMI %
Berg	1975	(3)	1612	2.5
Wisoff	1975	(37)	200	6.0
Hutchinson	1975	(18)	376	1.9
Cheanvechai	1974	(9)	545	4.0
Phillips	1977	(28)	3031	5.4
Kouchoukos	1977	(22)	710	7.9
Ullyot	1977	(35)	200	8.5
Assad-Morrell	1975	(2)	500	13.0

anesthesiologist, nursing personnel and ancillary personnel. The requirements for extended professional support with respect to renal failure, pulmonary problems, and neurologic problems are evident. Finally, the need for the broad facilities and services provided by a general hospital is suggested by the requirements for blood, hemodialysis, and respiratory support, etc.

Postcardiotomy syndrome is not a well defined entity. The syndrome which includes any combination of malaise, fever, pleurisy, anorexia, increased pain in relation to the operative incision, arrhythmias, usually atrial fibrillation is well recognized. We estimate that it occurs in excess of 30% of the patients and responds promptly to the administration of cortisone and less well to other anti-inflammatory agents. We do not have firm data to support our impressions regarding postcardiotomy syndrome and its treatment.

Intraaortic balloon counterpulsation is an exceedingly important life saving measure. Its utility and effectiveness have been extensively documented in the literature.[4,5,7]

We feel that intraaortic balloon counterpulsation should be initiated prior to cardiopulmonary bypass in patients with very poor left ventricular function based upon clinical, hemodynamic and angiographic data. It is also indicated, prior to cardiopulmonary bypass, in patients with preinfarction syndrome who have not responded well to medical measures. We believe it is indicated following cardiopulmonary bypass when there is demonstrably poor left ventricular function characterized by poor contractability of the ventricle, persistently elevated left atrial pressure, hypotension, and the need for large amounts of ionotrophic agents. Arrhythemia, if persistent and particularly when worsened by ionotrophic agents, is still another indication for IAPB.

The objective of aortocoronary bypass is complete revascularization without left ventricular damage. Unfortunately, objective improvement of the performance of the left ventricle does not often occur. Complete revascularization minimizes the incidence of perioperative infarction, impaired performance of the ventricle, and suboptimum results of surgery. A conviction prevails that all diseased vessels should be bypassed.

Recent reports, particularly that of Loop, et al.[19] encourage reoperation for recurrent angina. Two patients included in this group of 100 patients suggest a very satisfactory result from reoperation can be achieved provided the coronary arteriogram, and other pertinent data, demonstrate that bypass is feasible. The operative procedure proves to be more difficult and time consuming that the original procedure, and it is associated with a higher incidence of bleeding.

Very few question the effectiveness of aortocoronary bypass with respect to the alleviation of angina and a better quality of life in the majority of patients.

The weight of present day evidence suggests that the prognosis of patients with mainstem disease, double vessel and triple vessel disease is significantly compromised.[6,15,17,25] Notwithstanding a lively debate,[13,29,31,32] there is substantial evidence which supports the conviction, shared by the authors, that aortocoronary bypass does prolong life and lower the incidence of myocardial infarction in a significant number of patients.

The randomized VA study, recently reported by Murphy et al,[27] supports a certain skepticism. The validity and significance of this report has been questioned by many.

In conclusion, aorotocoronary bypass is a relatively safe procedure and it relieves unstable angina in 80-90% of patients. Because of the lack of suitable randomized studies, a commonplace problem in clinical medicine, it is still unresolved whether or not, or to what extent, aortocoronary bypass alters or betters the prognosis of patients with symptomatic coronary artery disease.

Aorotocoronary bypass for the asymptomatic patient with significant coronary artery disease is still open to question.

References available upon request because of space limitations.

Seminars in Chest Medicine

Pulmonary Stress Testing

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Stress testing has become a widely accepted procedure in the diagnosis of exercise-induced myocardial ischemia. Most physicians are familiar with this type of exercise testing which involves only EKG and blood pressure monitoring. Stress tests which also examine respiratory function are somewhat more complex but may be essential in the evaluation of exercise intolerance. Such studies are often important to: 1) establish the presence of exercise limitation and quantitate its severity, 2) aid in the differential diagnosis of exertion-limiting dyspnea, 3) clarify the pathophysiologic mechanisms underlying exercise limitation.

Understanding of the normal physiologic response to exercise is necessary in order to understand the mechanisms that impair exercise capacity in disease states. Normal work performance requires augmented ventilation, gas exchange and circulatory O_2 and CO_2 transport in order to meet the metabolic stress of exercise (Fig. 1). Minute ventilation normally rises during exercise due to increases in tidal volume at low work levels and due to increases in respiratory rate at higher levels of exercise (Fig. 2).[1] During exercise, physiologic dead space decreases due to a more even distribution of ventilation and perfusion throughout the lung (Fig. 3).[2] This results in a diminution of "wasted ventilation" which is sustained at all work levels, i.e., an increase in effective alveolar ventilation. The net result of increased alveolar ventilation during exercise is to maintain a stable arterial oxygen tension (p_aO_2) and prevent increases in arterial carbon dioxide tension (p_aCO_2) despite increased metabolic stress.

The cardiovascular system responds to higher energy demands during exercise by increasing cardiac output.[3] This results from increases in both stroke volume (SV)

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and heart rate (fH). Blood flow is preferentially redistributed to working muscles and away from other regions, such as, the renal and splanchnic circulations. As a result of these physiologic adaptions in the respiratory and circulatory systems, healthy individuals are capable of strenuous physical activity.

Exercise tolerance is diminished when a diseased cardiorespiratory system cannot meet the metabolic stress of exercise. By taxing the reserve capacity of the heart and lungs, exercise may elicit abnormalities not readily apparent at rest. Measurements made during exercise are therefore valuable in documenting exercise limitation arising from early pulmonary or cardiac dysfunction. Conversely, a normal response to exercise when symptoms have led to fear of early disease allows the clinician to more confidently advise and reassure his patient.

In patients with more advanced cardiopulmonary disease, stress testing provides an objective measurement of work performance. Resting studies unfortunately are not only unreliable in predicting work capacity, but do not identify the factors which limit exercise. Moreover, stress testing complements the patient's description of exercise-related symptoms by allowing observation of his reactions during exercise.

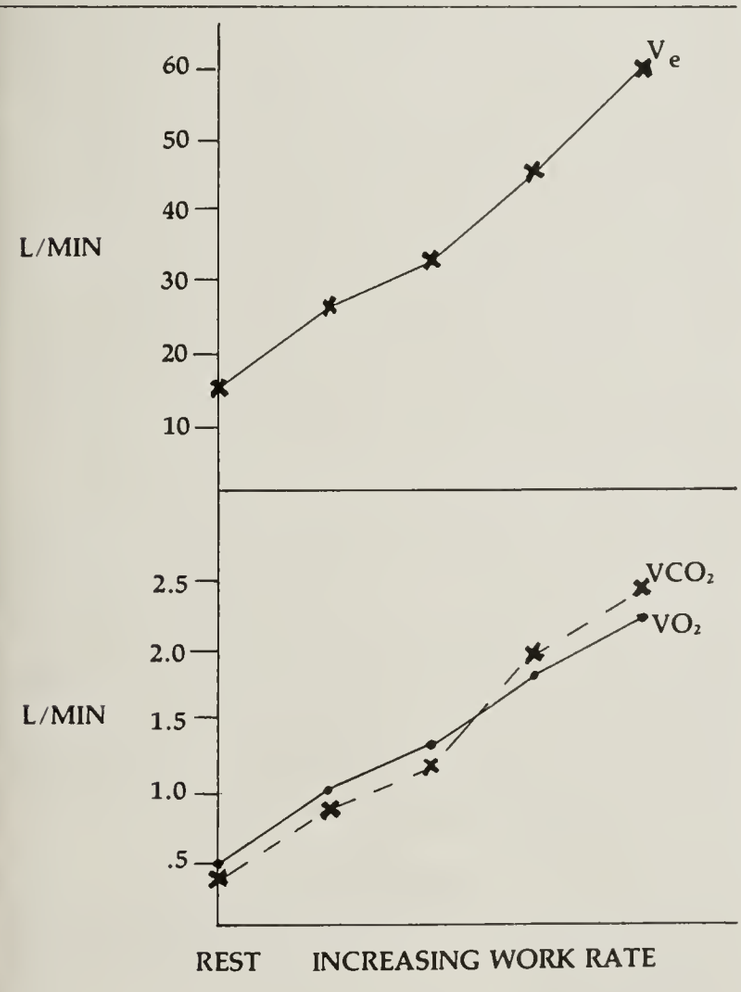


FIG. 1
Results of exercise study in a normal subject (P.D.) showing relationships between increasing work load and minute ventilation (V_e), carbon dioxide output (VCO_2) and oxygen consumption (VO_2).

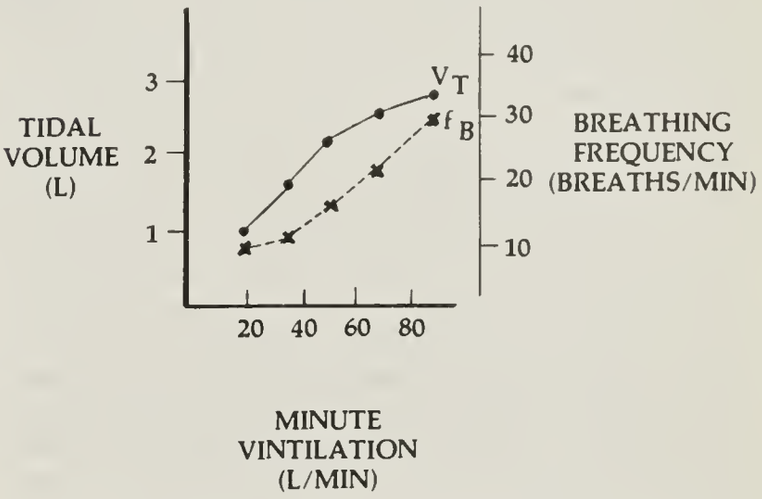


FIG. 2
Relationship between tidal volume (V_T), breathing frequency (f_B), and minute ventilation in a normal male (P.D.).

In a few conditions, such as exercise-included asthma and myocardial ischemia, response to exercise is diagnostic of a disease state. In many other significant cardiovascular and pulmonary problems an abnormal exercise pattern will be suggestive of a particular disease process, and will be important in quantitating the severity of that process. Specifically in chronic bronchitis, emphysema, cardiac valvular disease, pulmonary vascular disease, and infiltrative lung processes, the physiologic response to exercise will be extremely helpful in clarifying and quantitating the factors limiting work performance.

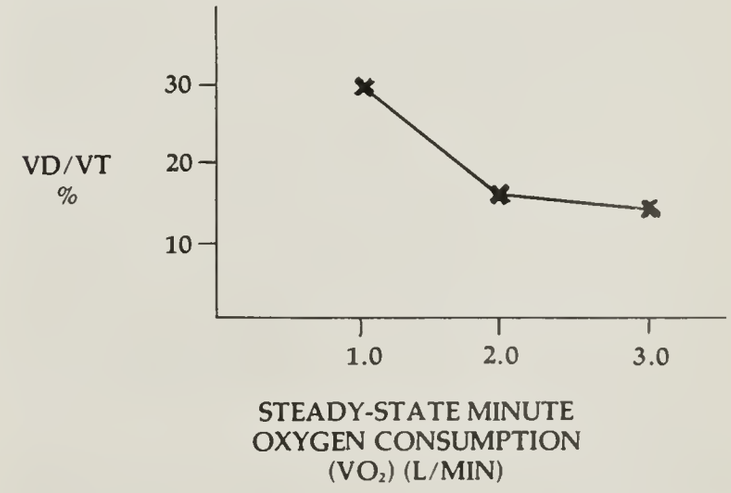


FIG. 3
Dead space/tidal volume ratios with increasing exercise in a normal adult subject (P.D., ♂).

A multiplicity of methods, varying in complexity and invasiveness, have been described to evaluate cardiopulmonary function during exercise. Stress testing should involve dynamic exercise employing large muscle groups, preferably at several work loads requiring increasing power output. Exercise tests involving small groups of muscles (i.e., hand grip) do not stress the cardiorespiratory system to any significant extent. Ideally, an exercise test should utilize the simplest and least invasive techniques that will yield the desired information. Based on these premises, Jones et. al.[4] have proposed a staging for exercise testing in order of increasing complexity and sophistication. The simplest study (Stage I) is a bloodless technique involving progressive exercise to symptom-limited maximum with measurements at each power output to include heart rate by EKG, blood pressure, ventilation, tidal volume and respiratory rate. Subsequent stages require more elaborate measurements, with the most complex (Stage IV) including data on right heart pressures obtained via Swan-Ganz technique. An abnormal response to a Stage I test obviates the need for more complex testing, since these are unlikely to add further useful information.

The following examples will illustrate the type of abnormalities encountered during exercise testing in patients with cardiopulmonary dysfunction.

EXAMPLE I

A 36-year-old housewife with angiographically-demonstrated pulmonary emboli continued to complain of severe breathlessness with exertion one year following the acute episode. Repeated perfusion lung scans revealed residual defects, but no evidence of recurrent embolization. Pulmonary function studies and arterial blood gases were normal at rest. A progressive exercise study was performed; it showed significant decrease in paO_2 and widening of the alveolar-arterial oxygen difference at increasing work loads. The VD/VT rose from a normal of .35 at rest to over twice the predicted value at maximal exercise. These findings reflect ventilation-perfusion imbalances secondary to pulmonary vase-occlusive disease, i.e., the increased ventilation of nonperfused alveoli which occurs with exercise leads to an increase in "wasted ventilation." In addition, the decreased volume of the pulmonary capillary bed in vaso-occlusive disorders shortens erythrocyte time through the capillary bed. This time is further shortened as cardiac output increases during exercise. As a consequence, the red blood cells are not exposed to the lung's air interface for a sufficient time to achieve optimal oxygen loading. This example illustrates that normal pulmonary function tests and arterial blood gases at rest do not rule out the presence of significant exercise-limiting functional disturbances 2^0 to pulmonary vascular disorders.

EXAMPLE II

A 47-year-old rancher with long-standing rheumatoid arthritis complained of chest discomfort and dyspnea with moderate exertion for approximately one year. Chest x-ray, EKG, spirometry and lung volumes were all within normal limits; single-breath carbon monoxide diffusing capacity, however, was found to be diminished at rest. Exercise studies at moderate work levels revealed a pattern compatible with early pulmonary restriction. Increases in minute ventilation with exercise were achieved by rapid respiratory rates, rather than by increasing tidal volumes, even at relatively low work levels. This occurs in patients with restrictive pulmonary disease because excessive inspiratory effort is needed to distend their noncompliant lungs. Subsequent transbronchial lung biopsy in this patient revealed a pattern of interstitial fibrosis, presumably 2° to his collagen vascular disease.

EXAMPLE III

A 52-year-old white female with known mitral stenosis secondary to rheumatic heart disease and history of 1 ppd cigarettes x 20 years was referred for assessment of gradually increasing exertional dyspnea. Spirometric studies were all normal for the patient's age and size. Progressive exercise testing showed an elevated heart rate at all work levels with relatively normal ventilatory studies. Although her maximum power output was only 50% of predicted, she reached maximum predicted cardiac frequency at this workload. Thus, she had reached her cardiovascular limitation to exercise. This exercise data strongly suggests an inadequate cardiovascular adaptation to exercise resulting from a diminished left ventricular stroke volume due to mitral stenosis.

In summary, cardiopulmonary exercise studies when properly performed and interpreted can be an important clinical tool in the evaluation of exercise limitation. Although the risks of stress testing are relatively small when patients are adequately screened and some simple precautions are observed, every exercise laboratory must have adequate, accessible facilities for resuscitation. Situations where the risks of exercise testing are generally felt to outweigh its value include, among others, severe aortic stenosis, recent myocardial infarction (within the prior 3 months), and uncontrolled cardiac arrhythmia or heart failure. A physician familiar with the procedure must always review the patient's clinical history and physical findings prior to testing. This physician should also supervise the test and be alert for untoward effects of the procedure, such as, dizziness or faintness, cyanosis, decrease in systolic BP greater than 25 mm Hg, or frequent PVCs. Finally, meaningful interpretation of data obtained requires knowledge of both normal and abnormal cardiopulmonary response to exercise.

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Seminars in Endocrinology and Metabolism

Multiple Endocrine Adenomas I & II

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To understand multiple endocrine adenomas (MEA) and their groupings, one must start with embryology. Experimentally, the neural crests of Japanese quail embryos which have large individual cells, have been grafted into chick embryos which had their neural crests removed. These neural crest cells which are pluripotent, (i.e. They can form various types of cells.), immediately begin to migrate out of the neural crest region. As they migrate out, they are influenced by the environment into which they find themselves to lose their pluripotentiality and differentiate into more specialized cells.[1] These cells will ultimately form the autonomic nervous system, the adrenal medulla, the ultimo-branchial bodies, Type I cells of the carotid bodies, endocrine cells of the pancreas, entercholinergic ganglia (e.g. Auerbach's plexus), cholinergic ganglia of the pancreas and some cells of the anterior pituitary, bronchi and gastro-intestinal tract.[2]

Besides their neural crest origin, all these cells have in common an ability to synthesize aromatic amines or polypeptides or both. They also have the capability to take up, decarboxylate and store amine precursors. For this reason they are called APUD cells (Amine Precursors Uptake and Decarboxylation). A tumor of this system is thus called an Apudoma.[2]

When APUD cells become oncogenic, they may regain part of their pluripotentiality and produce hormones they would not normally produce i.e. ectopic hormones. A prototype is the bronchial carcinoid which can make ACTH, insulin, growth hormones, glucagon, calcitonin, prolactin, anti-diuretic hormone, catecholamines.

There are two main types of familial multiple endocrine adenomatosis. Although there have been a few scattered reports of patients having tumors from both types, in family kindreds the types appear to run exclusive of one another.

Type I (Werner's Syndrome) includes abnormalities of the pituitary gland, parathyroid gland and endocrine pancreas. The pituitary may display hyperplasia or adenoma formation. Of the pituitary adenomas, 45% are chromophobes, 25% are acidophilic i.e. they are producing growth hormone leading to acromegaly. The parathyroid glands may show either hyperplasia or adenomatosis.[3]

The pancreas usually demonstrates an islet cell adenoma, frequently multiple. The secretion of this tumor may be insulin, glucagon, gastrin or any combination. The reason for this array of hormones is found by looking at the anatomy of the islet. By histochemical staining, four distinct cell types have been identified: the cell which normally produces glucagon, the B cell which normally produces insulin, the D cell which produces somatostatin (growth-hormone inhibitory substance) and VIP (vasoactive intestinal peptide) and the P cell — a minor cell type which produces PP (pancreatic polypeptide — a hormone increased by eating which in turn can increase insulin release).[4]

Since the D cells also produce VIP, a few patients may have incresed levels of VIP and demonstrate a causually related syndrome of watery diarrhea — referred to as the Verner - Morrison syndrome.

50% of patients with Type I MEA will demonstrate peptic ulcer disease (2/3 of these patients will have the Zollinger-Ellison syndrome); 88% will have parathyroid adenomas; 81% have pancreatic adenomas; 65% have pituitary adenomas. Occasionally an adrenal or thyroid adenoma may be demonstrated.[5]

If all patients with the Zollinger-Ellison syndrome are assessed, 30% will be identified as having an MEA Type I. Indeed, it has been postulated that the Zollinger-Ellison phenotype is a variant of the same mutant gene. A little confusion arises since the Zollinger-Ellison syndrome in and of itself can lead to parathyroid hyperplasia. The proposed mechanism is as follows: ↑ gastrin → ↑ calcitonin → slight ↓ calcium → ↑ parathyroid hormone. With chronic stimulation of parathyroid hormone, hyperplasia can occur. Since the basis of the hyperplasia is the increased gastrin, most cases of hypercalcemia will remit if the tumor can be removed.

The usual presentation of patients with MEA Type I is with peptic ulceration. The ulcers are florid, multiple, in

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odd places and recalcitrant to treatment.

The pituitary adenoma may make its presence known by galactorrhea in the case of a chromophilic adenoma or by acromegaly.

Those with an insulinoma often have severe fasting hypoglycemia. Those with a glucagonoma may have a stomatitis, vaginitis, raw tongue and a peculiar desquamating skin rash. Because of increased glucagon, mild diabetes is often present as well as a chronic disease type of anemia.

Symptoms of parathyroid disease are usually lacking although bone pain and lower extremity muscle weakness are sometimes found.

Despite possible presentations, the usual symptoms are gastrointestinal. This is not surprising since a full 50% of these patients will die of peptic ulcer complications.

Treatment of both MEA Type I and II is surgical. Primary removal of the hyperplasia or adenoma is considered optimal, if possible. If there is an inability to find the tumor or if metastases are present, a total gastrectomy is often done in patients with gastrinomas. Recent reports of the use of Cimetadine (an H_1 histamine blocker) to treat non-resectable gastrinomas have appeared. However, the authors have cautioned its use for only those patients who cannot tolerate gastrectomy.[6]

On patients with both gastrinoma and hypercalcemia, recommendation is for removal of the gastrinoma first with the hope the hypercalcemia will ameliorate when the stimulus of gastrin is removed.

Type II MEA can be further divided into two subtypes: II a (also called Sipple's syndrome) includes medullary thyroid carcinoma and pheochromocytoma. Frequently there is associated parathyroid disease and rarely an ACTH producting tumor of the pituitary. Type II b also includes medullary thyroid carcinoma and pheochromocytoma. In addition those patients have a Marfanoid habitus, mucosal neuromas, as well as von-Recklinghausen's disease and hyperplastic corneal nerves. They rarely have hyperparathyroidism.

The natual history of medullary thyroid carcinoma seems to be C-cell hyperplasia progressing to a solid nodule or goiter, often multicentric. It may resemble other types of thyroid carcinoma, e.g. the spindle cell carcinoma, but is distinguishable because it uniquely stains from amyloid. It is a late metastisizing tumor which first may involve the rest of the gland and then reach the regional lymph nodes. From there it often spreads, via the bloodstream, to lung, bone and liver.

Since the origin of medullary carcinoma is the C cell (parafollicular cell), this carcinoma will produce increased amounts of calcitonin. Indeed, the diagnostic test for screening families with Type IIa MEA is to measure levels of calcitonin. A high level of calcitonin in the blood is presumptive evidence that the individual either has C cell hyperplasia or medullary carcinoma. Previously, thyroid vein sampling was also done with a step-up in calcitonin levels pinpointing the location of the C cell hyperplasia.[8]

In the presence of metastases, treatment besides thyroidectomy, is Adriomycin. Response to therapy is stated to be 40%.[9]

Within the past few years, it has also been found that the medullary thyroid carcinoma, presumably because of ancestory from the APUD system, is capable of producing ACTH giving a Cushing's syndrome and of also producing serotonin with a carcinoid like picture.

Pheochromocytomas are generally symptomatic lesions. Most typically there is hypertension either sustained or paroxysmal. However, other manifestations include episodic or continuous sweating with profound flushing or blanching, postural hypotension (with excessive amounts of catecholamines acting as ganglionic blockers), myocarditis.

Physical examination may reveal profuse sweating, cafe-au-lait spots, neurofibromas, port wine spots or telangiectasia over the conjunctivae. On occasion, applying pressure to the abdomen may induce a rise in blood pressure or increased sweating.[10] Screening for a pheochromocytoma is done with a 24 hour urine collection for catecholamines and metanephrines (the degradation product of catecholamines). Although a number of provacative tests have been proposed for demonstrating a pheochromocytoma. These are not advised since a tumor will generally manifest itself if it is present.[11]

Treatment is surgical extirpation, with the surgeon exploring both adrenals and the organs of Zuckerkandl since the tumor may be multiple. Preparation for surgery is accomplished using an α blocking agent such as phentolamine or dibenzyline to prevent hypertensive crises during exploration and removal. Alternatively, dibenzyline can be used chronically in patients with metastases or those considered too great a surgical risk.

In both types of MEA, an emphasis must be placed in examining all family members yearly after the age of 10 years for early detection of any of the abnormalities. Genetic counseling is also suggested.

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Seminars in Gastroenterology and Liver Disease

Melena and Occult Blood in the Stool

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Melena

Melena is defined as the passage of black tarry stools from the rectum. In over 90% of instances, it is due to a bleeding lesion above the papilla of Vater.[1] The stool is coal black, sticky, shiny, foul smelling, and of a tarry consistency. It has been estimated that 50-60 cc. of blood must be in the stool for melena to be present.[2] The site, amount, and rapidity of bleeding, and the rate of intestinal transit are all important in determining whether melena will occur.

It has been estimated that blood must be present for 8 hours in the intestinal tract to turn to a black color. The black color is believed to be related to the presence of porphyrin pigments derived from the heme portion of hemoglobin. Although the usual causes of melena are such bleeding lesions as duodenal and gastric ulcers, gastritis, esophagitis, and esophageal varices, occasionally it can be caused by bleeding points in the distal small bowel, as the following case report will attest. Rarely, a slowly bleeding lesion in the cecum or even the right colon can result in melena.

A 43-year-old white male patient presented to the emergency room with a two-day history of melena. There was an associated gradual onset of weakness which culminated in a single syncopal episode on the day of admission. He denied any recent alcohol use, but he did give a history of ingestion of multiple aspirin and Motrin tablets for arthritis on the previous day. He had undergone an oversewing of a bleeding duodenal ulcer at 19 years of age, but had had no ulcer symptoms since then.

Physical examination on admission was essentially negative except for frank melena confirmed by rectal examination. Admission laboratory findings showed a hemoglobin of 6.8 gm% with a hematocrit of 19.5%. SMA-12, electrolytes, creatinine, PT and P.T.T. were all within normal limits. Chest x-ray and KUB were also normal.

Following his admission he underwent immediate esophagogastroscopy which revealed no abnormalities and no bleeding site. Following administration of four units of packed cells, the hemoglobin increased to 9.1 and the hematocrit to 29%. His vital signs were stable and he ceased to pass any more tarry stools. On the next day attempts were made to do a barium enema but this procedure had to be abandoned due to the sudden onset of tachycardia, hypotension, dizziness and the passage of three to four bulky melanotic stools. The hemoglobin dropped to 8.2 and the hematocrit to 20. Attempts at performing arteriography were unsuccessful. The patient then underwent exploratory laparotomy at which time a four centimeter intramural mass was found in the distal ileum, two feet from the ileocecal valve. This mass was excised. Intra-operative pathology consultation proved this mass to be ectopic spleen with focal necrosis, ulceration and hemorrhage into the intestinal lumen. Postoperatively, follow-up UGI series with small bowel follow through and barium enema showed no other abnormalities. The patient to date has had no further bleeding episodes and has made an uneventful recovery.

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Occult Blood

Frequently, blood is present in the stools in amounts insufficient to produce a black color. This is termed occult bleeding, and it can be tested for by one of several simple and rapid screening methods. All of these tests are based on the production of a blue color by the oxidation of a phenolic compound to a quinone structure with an intramolecular reaction. Orthotolidine (Hematest) and gum guaiac (guaiac and Hemoccult) are the sources of the color production mediated by hydrogen peroxide as the oxidizing agent. Peroxidase activity (important in catalyzing this reaction) is provided by hemoglobin, but can also be caused by other peroxidases in red meat, fish and vegetables, and by catalase.[3]

The Hematest reaction is performed by placing the tablet on a stool specimen and then allowing several drops of the testing solution to flow over the tablet and onto the paper. A blue color developing in 1 minute is considered a strongly positive reaction, in 2 minutes weakly positive, and no blue color at two minutes a negative reaction. The guaiac reaction is performed by dripping a few drops of glacial acetic acid on the stool, followed by 2 drops of guaiac solution (gum guaiac in 95% ethanol), followed by several drops of H_2O_2. A blue color appearing within 30 seconds is a strongly positive reaction, in 1 minute a weakly positive one, and no blue color at the end of 1 minute is a negative reaction. The Hemoccult test is done by smearing a small amount of stool on the prepared card and then placing 2-3 drops of the supplied solution on the stool. At the end of 30 seconds an intense blue color is a strongly positive reaction, a lighter blue color or one with a greenish tinge is weakly positive, and no blue color at the end of 30 seconds is a negative test.

Both the Hematest and guaiac reactions are highly sensitive, and the Hemoccult test less so, about ¼ as sensitive as the other two.[4] As little as 2-3 ml. of blood in the stool may give a positive reaction by these methods. However, as 2 ml. of blood loss into the stool is accepted as normal, the Hematest reaction was shown to result in 76% falsely positive tests, the guaiac 72%, and the Hemoccult 12%, in patients with potentially bleeding lesions.[4] When healthy medical students were fed Cr^{51} labeled red blood cells, the Hematest reaction resulted in 27% false positives, the guaiac reaction nearly 50%, and the Hemoccult 11% as judged by the concentration of the Cr^{51} label in the stool.[5]

The high percentage of false positive reactions by the Hematest and guaiac reactions make these unreliable indicators of actual occult bleeding. Eliminating red meat and vegetables from the diet improves the number of false positives, but does not eliminate them.[6] The Hematest reaction resulted in 53% false negative tests when significant quantities of blood were present in the stool, and its continued employment as a screening test has been seriously questioned.[4] A similar high false negative reaction rate with guaiac solution, and the world-wide scarcity of the gum guaiac reagent, make this test similarly less than ideal for clinical situations.[4]

Although the Hemoccult reaction is less sensitive than the Hematest and guaiac tests, most of the false positive tests are weakly positive, and when tested serially, the majority of the tests are negative when no blood is present in the stool. A 20% false negative reaction with Hemoccult is largely overcome by testing at least 3 stool samples.[4] On a vegetable containing, no meat diet, a positive Hemoccult reaction on at least one of 3 stools was shown to be associated with a colonic lesion in 80% of patients.[7]

Supplemental iron rarely will produce a false positive Hemoccult reaction. Barium in the stool does not effect this test, and the employment of laxatives reduces the rate of false positives.[4] Storage of the Hemoccult slide at room temperature diminishes the detection of a positive reaction but this effect is overcome by putting a few drops of water on the slide prior to adding the indicator solution.

Although fish, chicken and vegetable peroxidases can catalyze the Hemoccult reaction and result in a falsely positive test, these are inactivated by passage through the intestinal tract, so that only red meat in the diet serves as a practical source of potential false positive reactions.

Summary

Unless iron, licorice or bismuth containing substances are ingested, a black stool should strongly raise the suspicion that melena is present. Of the chemical tests available for detecting blood in the stool, the Hemoccult test may be the most useful and dependable one if 3 or more stool samples are checked with the patient on a red meat-free diet. A negative series of tests is reassuring, and one or more positive ones should result in further investigation.

The benzidene test, which is more sensitive than any of the above, is no longer available as its manufacture was associated with carcinogenic hazards. The ideal test for screening for blood in the stool, i.e., always positive when blood is present in the stool and always negative when no blood is in the stool, is not yet available.

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Obstetrics and Gynecology

Sodium In Pregnancy Part II

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Donald J. Ziehm, M.D., Editor

Should the use of sodium be forbidden during pregnancy? If a normal gravida markedly decreases dietary sodium, serum sodium will also decrease. Recognized by the macula densa of the kidney, the normal renal processes are thereby activated-secretion of renin, angiotensin I, antiotensin II and aldosterone. The end result is sodium retention. Thus, limiting sodium intake does little more than stimulate normal renal mechanisms. The average gravida must retain considerable amounts of sodium to provide for her tissue requirements, extracellular and intravascular fluid requirements, and the tissue needs of the reproductive weight mass.

What takes place in toxemia? According to current literature, the earliest detectable change in pre-eclampsia is an undue sensitivity of arterioles to angiotensin II, the catecholamines, and cold. This evolves much earlier than any unusual tendency for storage of water. Plausible explanations for water retention in pre-eclampsia exist without having to invoke abnormal renal handling of sodium. If arteriolar muscular tone increases in organs and tissues, the forward progress of blood thru capillaries is limited. If this becomes widespread, stasis of blood within the capillaries occurs, resulting in decreased reabsorption of the water of aerobic metabolism. Consequently, edema will develop on this basis without any deficiency in renal handling of sodium. Total body sodium is increased in toxemia. Hypotonic water can not accumulate. Therefore the amount of sodium contained in the water of aerobic metabolism will increase (secondary to decreased propulsive force through the capillaries and decreased takeup of water metabolism). Generalized edema develops. Edema may be present in all areas of low tissue tension-skin of the fingers, periorbitals, the skin over the manubrium, the skin of the face.

Despite the fact that circulating angiotensin II is significantly increased during pregnancy, there is generally a decrease in mean blood pressure as well as considerable arteriolar vasodilation. For some reason the vasoconstrictor action of angiotensin II is blocked in normal pregnancy. In pre-eclampsia something emerges, develops, is elaborated, or fails to be elaborated, that alters the sensitivity of the pre-capillary arterioles to angiotensin II, resulting in edema. As a concomitant of vaso-spasm, arterial blood pressure is increased. Blood pressure is a function of the total expulsive force of the left ventricle, aortic rebound and peripheral resistance. If a sufficient number of pre-capillary arterioles develop spasm, blood pressure will increase. A mechanism similar to that which produces albumin in the urine occurs. Vasoconstriction in the arterioles supplying the glomerulus produces stasis, hypoxia, and apparently a dissolution in the continuity in the walls of the glomerulus. The intercellular cement presumably loses its integrity and albumin molecules

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escape. Consequently, at least speculatively, all of the events leading to pre-eclampsia can be explained without abnormal handling of sodium.

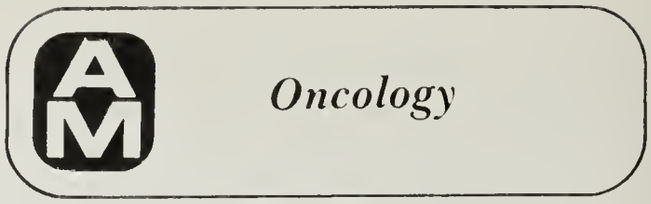
Dependent edema, it should be noted, is not related to sodium retention. The normal gravida often has dependent edema, secondary to uterine pressure on pelvic veins. Venous back pressure is transmitted to the capillaries; the propulsive force through the capillaries is thereby limited. Again, the water of metabolism which is constantly formed in aerobic metabolism, accumulates. Water is picked up from the extracellular fluid compartment of the capillaries because of osmotic force, basically by albumin. Pick-up of water is limited whenever the progress of albumin through the capillaries is impeded.

If blood remains static, the capillary attraction (attraction of water across the capillary walls) will ultimately cease when equilibrium is established. Therefore, the edema resulting from pressure upon the pelvic vein has nothing to do with sodium ion abnormalities. Simply putting a gravida at bed-rest with elevation of the legs will cause the edema to disappear. Edema of pre-eclampsia may also be relieved by bed-rest. A large number of toxemic patients will experience a decrease in blood pressure, in addition to a considerable diuresis, without the use of a low sodium diet or diuretics, simply due to the vascular changes that occur with bed-rest.

Are diuretics indicated in the management of toxemia? Is the sodium ion really important in genuine pre-eclampsia, assuming that all of the classical symptoms are present? Those who argue in favor of diuretics do so from the erroneous assumption that the kidney is processing sodium in some abnormal fashion. In addition, the supposition persists that convulsions are related to cerebral edema. There is no evidence for this at all. Older clinicians remember the "dry toxemics"-women with little or no edema who still convulsed. They will recall the infusion of 50% D/W to mobilize edema fluid from the brain to prevent convulsions.

Admittedly, with limited sodium intake a diuresis may occur. Also if diuretics are prescribed (specifically the Thiazide diuretics which act by limiting tubular reabsorption of sodium) the patient will also diurese. But the clinical course of toxema is usually not altered by this treatment.

Controlled experiments on human gravidas are difficult to perform. The best one can do is to study the mechanisms carefully and attempt to arrive at a philosophic position from which to conduct clinical activity. To repeat, until now (for the last forty or more years) the presumption has been prevalent that the sodium ion was harmful in pregnancy, and salt intake was limited. Current evidence reveals no physiologic or pathologic basis for this practice. It is not necessary to withhold salt from a pregnant woman. Withholding sodium from a toxemic is not a critical or desirable element of the management. Edema is not critically implicated in the development of convulsions or of any of the other important problems relating to toxemia.



Detection of Adriamycin Cardiotoxicity by Echocardiography:

Gordon A. Ewy, M.D.
Stephen E. Jones, M.D.
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Editors:
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Robert H. Thoeny, M.D.

The anthracycline antibiotic adriamycin is an important addition to the oncologist's drug armamentarium as it is highly effective when given singly or in combination against a wide variety of human cancers. The major dose-limiting side effect of this agent is the development of a cardiomyopathy.

Adriamycin cardiac toxicity shares the clinical features of other congestive cardiomyopathies: the insidious onset of symptoms and signs of congestive heart failure due to a poorly functioning myocardium. Rarely arrhythmias may occur. Often the patient may symptomatically improve with standard modes of therapy (sodium restriction, bedrest, diuretics, digitalis), but the underlying cardiomyopathy is generally felt to be irreversible and even fatal. Currently the only established means of avoiding adriamycin cardiac toxicity is by limiting the total lifetime dose of the drug to less than 550 mg/m^2.

In a previous article in *Arizona Medicine* we reviewed the use of non-invasive techniques to measure heart function in order to detect subclinical cardiac damage from adriamycin before the development of overt heart failure.[1] The measurement of "systolic time intervals"

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from phonocardiograms and the calculation of frontal plane limb lead voltage from electrocardiograms are examples of noninvasive techniques which have shown promise.[2,3]

During the course of evaluating patients with adriamycin cardiomyopathy we were able to demonstrate the development of abnormal ventricular function by another noninvasive technique, echocardiography. In this month's column we have reviewed our experience to date with this technique.

PATIENTS & METHODS

Since May 1974 echocardiograms have been recorded on adult patients with biopsy-proven cancer being considered for, or already undergoing, adriamycin treatment. The initial patients studied had had at least one course of adriamycin. Subsequently, all patients considered for therapy had control or baseline studies (zero dose), and if adriamycin therapy was initiated serial studies were performed at three-month intervals. The mean age of the patients studied was 55 years. Forty-five percent of the patients had breast cancer, 12% had gynecologic cancer, 10% had lung cancer, and the remainder had a variety of other solid tumors or hematologic malignancies.

The echocardiograms were obtained with a commercial echocardiograph (Echoline 21) having a repetition rate of 1,000 pulses per second utilizing a 2.25 MHz transducer with a 13-mm crystal. The echocardiograms were recorded on a Honeywell 1856 strip chart recorder with simultaneous lead II electrocardiograms. The patients were examined in the supine or slightly left lateral position with the transducer at the left sternal border in the interspace where the cardiac echos were best seen. The cardiac echos were continuously recorded as the transducer was tilted slowly through an arc, with the ultrasonic beam traveling from the area of the aortic valve to the left ventricle. The echocardiographic left ventricular measurements were made where the left ventricle was best seen in the scan at the area of the tip of the mitral valve and/or chordae.

Figure 1 illustrates a normal left ventricular echocardiogram and demonstrates how the measurements were made. Left ventricular internal diameters were taken from the left side of the interventricular septum to the posterior left ventricular endocardium during both end-diastole (Dd) and end-systole (Ds). The left ventricular diastolic dimension was made at the QRS complex and the systolic measurements were made on a line extending vertically from the most anterior portion of the posterior left ventricular endocardial surface. These measurements were used to calculate the echocardiographic ejection fraction (EF) by the cubed method:

$$EF = \frac{(Dd)^3 - (Ds)^3}{(Dd)^3}$$

Figure 2 illustrates the development of an abnormal echocardiogram in a patient receiving adriamycin and thoracic irradiation.

The clinical features and echocardiographic data were coded and analyzed with the Statistical Package for the Social Sciences on a CDC 6400 computer at the University of Arizona. Initial bivariate analysis revealed that

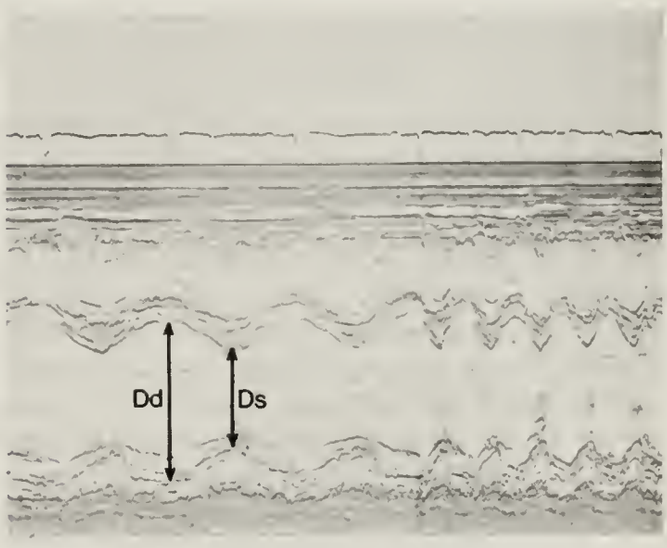


FIGURE 1

Normal echocardiogram. The arrows indicate the distance from the endocardium of the left ventricular side of the intraventricular septum to the endocardium of the posterior wall of the left ventricle. The left ventricular diastolic diameter (Dd) is measured at the onset of the QRS in the reference electrocardiogram. The left ventricular systolic diameter (Ds) is measured vertically from the most anterior excursion of the posterior endocardium. The vertical dots on the left side of the photograph indicate a distance of 1 cm. Echos from chordae are seen in the mid to lower portion of the left ventricular cavity. The echo ejection fraction (EF) was 0.76 (normal).

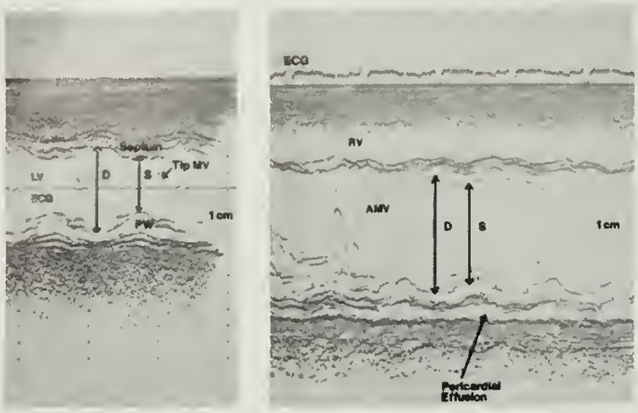


FIGURE 2

Echocardiogram of a 52-year-old female with carcinoma of the breast with metastatic lesions. She had received 494 mg/m² of adriamycin between 10/16/73 and 8/20/74. She had also received radiation therapy to a tumor mass in the right hilar area.

LEFT: Echocardiogram obtained on 8/12/74. Left ventricular diastolic diameter (Dd) was 49 mm, left ventricular systolic diameter (Ds) was 33 mm, and the echocardiographic ejection fraction was 0.69 (normal). LV = left ventricle; ECG = electrocardiogram; PW = pericardial wall; MV = mitral valve.

RIGHT: Echocardiogram obtained 11/1/74 after 494 mg/m² of adriamycin. Left ventricular diastolic diameter (Dd) was 53 mm and left ventricular systolic diameter was 45 mm. The echocardiographic ejection fraction was 0.39 (abnormally low). A pericardial effusion was present. RV = right ventricle; AMV = anterior mitral valve.

total adriamycin dose, sex, thoracic irradiation, and cardiac risk factors (i.e., angina, hypertension, etc.) appeared to affect some of the echocardiographic parameters. These were then subjected to a statistical regression technique that permits simultaneous assessment of separate and interacting effects of several factors.

RESULTS

It was possible to obtain technically satisfactory echocardiograms in 62% of the adult patients with cancer. In 38% of adult patients it was impossible to obtain technically satisfactory tracings due to obesity, chest wall configuration and other individual factors. In patients with satisfactory echocardiograms there was a decrease in ejection fraction in some patients receiving high doses of adriamycin. Despite the use of several statistical methods, no consistent pattern of abnormality on echocardiogram was observed in patients receiving less than 450 mg/m² of adriamycin. Therefore, this report describes a comparison of echocardiographic findings in 185 patients studied prior to therapy (zero dose) with the results obtained from 27 patients who had received more than 450 mg/m² of adriamycin.

Table I presents the echocardiographic measurements grouped according to the patient's sex, dose of adriamycin, and the presence or absence of thoracic irradiation. Group I patients had neither a history of cardiac disease nor thoracic irradiation. Group II patients had received, or were receiving, thoracic irradiation. There was no significant difference in echocardiographic measurements of cancer patients without a history of cardiac disease or thoracic irradiation, irrespective of adriamycin dosage (Table I, Group I). However, there was a significant increase in left ventricular systolic diameter corrected for body surface area, Ds(BSA), and a significant decrease in the echocardiographically derived ejection fraction in male patients who had previous or concomitant thoracic irradiation and had received more than 450 mg/m² of adriamycin (Table I, Group II) compared with those who had not received thoracic irradiation. These same trends were also apparent for women who received thoracic irradiation (Table I), but the differences are not yet significant.

COMMENT

Single crystal echocardiography is of limited value in the assessment of ventricular function in patients with ischemic heart disease with segmental left ventricular wall motion abnormalities. In patients with symmetrically contracting left ventricular chambers, single crystal echo measurement of left ventricular systolic and diastolic internal diameter provides a reasonable estimate of ventricular volume. The echocardiogram has proven useful in assessing ventricular function in idiopathic congestive cardiomyopathy.[4] Since adriamycin cardiomyopathy appears to be secondary to diffuse myocardial damage, one would predict that echocardiography would be able to detect alterations in ventricular volume and function. This study demonstrates that the echocardiogram can indeed be used to assess cardiac function in adult patients receiving adriamycin.

A recent report indicates that echocardiography is useful for the *serial* assessment of ventricular function in children receiving adriamycin.[5] However, we were able to obtain satisfactory tracings in only 62% of adult patients with cancer. Thus, echocardiography will probably not be suitable for following all adult patients receiving adriamycin, but might prove of value in serial studies of patients from whom satisfactory echocardiograms can be obtained. If one is to determine the most sensitive and reliable method of following patients who are receiving adriamycin therapy, it will be necessary to directly compare echocardiography with the other invasive and noninvasive techniques which have been previously employed.[1] This analysis is presently underway in the patients being studied at the University of Arizona.

This study also provides insight into the mechanical sequence in which decreased cardiac function develops in patients with adriamycin-induced cardiomyopathy. For example, in the male patients who had received thoracic irradiation (Group II), the Ds/BSA increased from 24 ± 4 mm at zero dose to 37 ± 11 mm at high adriamycin dose and the Dd/BSA increased from 38 ± 6 mm at zero dose to 44 ± 8 mm at high adriamycin dose (Table I). The systolic diameter increased by a mean of 13 mm, while diastolic diameter increased by a mean of 6 mm. This may indicate that the earliest abnormality in adriamycin cardiomyopathy is a decreased systolic contraction rather than significant cardiac dilatation. It is this loss of systolic contractility that results in a decreased ejection fraction.

TABLE I

ECHOCARDIOGRAPHIC FINDINGS IN PATIENTS WITH CANCER
PRIOR TO ADRIAMYCIN THERAPY AND AFTER 450 MG/M² (TOTAL DOSE)

Total Adriamycin Dose (mg/m²)	Echocardiographic Parameter*	Mean Measurement (mm) ± 1 SD (No. of Patients)† GROUP I No Thoracic Irradiation Men	GROUP I No Thoracic Irradiation Women	GROUP II Thoracic Irradiation Men	GROUP II Thoracic Irradiation Women
zero	Ds/BSA	25 ± 3 (38)	25 ± 4 (97)	24 ± 4** (27)	24 ± 4 (23)
⩾ 450	Ds/BSA	27 ± 5 (5)	30 ± 8 (11)	37 ± 11** (6)	34 ± 8 (5)
zero	Dd/BSA	37 ± 4 (38)	39 ± 5 (97)	38 ± 6 (27)	38 ± 6 (23)
⩾ 450	Dd/BSA	38 ± 3 (5)	40 ± 8 (11)	44 ± 8 (6)	42 ± 5 (5)
zero	EF	.68 ± .10 (38)	.73 ± .09 (97)	.75 ± .08** (27)	.75 ± .09 (23)
⩾ 450	EF	.63 ± .13 (5)	.59 ± .17 (11)	.44 ± .20** (6)	.48 ± .19 (5)

* Ds/BSA = Left ventricular interval diameter at end systole in mm per m² of body surface area.

Dd/BSA = Left ventricular interval diameter at end diastole in mm per m² of body surface area.

EF = Echocardiographic ejection fraction.

** $p = .001$

† Patients without prior or concurrent thoracic irradiation or history of cardiac disease (Group I) vs. patients with prior or concurrent thoracic irradiation (Group II).

Previous reports have suggested that thoracic irradiation may contribute to the development of adriamycin cardiotoxicity.[1] This echocardiographic study demonstrates decreased ventricular function in patients who have received high-dose adriamycin and radiation to the thorax and supports the view that thoracic irradiation is a factor for the development of adriamycin cardiotoxicity. Fewer dose-related abnormalities in ventricular function were apparent by echocardiography in patients without prior or concurrent thoracic irradiation and without preexisting hypertension, angina, myocardial infarction or valvular heart disease (Table I, Group I). These findings support the recommendation of Minow *et al.* that the total dose of adriamycin be restricted to 450 mg/m² in patients who have thoracic irradiation.[6]

SUMMARY

Echocardiography was employed to evaluate ventricular function in adult cancer patients being considered for, or undergoing, adriamycin therapy. The echocardiograms of 185 patients with a variety of cancers who had not yet received chemotherapy were compared with the echocardiograms of 27 patients who had received more than 450 mg/m² of adriamycin. In male patients who had received a total adriamycin dose of ⩾ 450 mg/m² and thoracic irradiation there was a significant decrease in the echocardiographic ejection fraction when compared to control patients (zero adriamycin dose). In the group of patients who received high doses of adriamycin alone, ejection fraction was also less than that of control patients (zero adriamycin dose), but the difference was not statistically significant. Hence, thoracic irradiation was identified as a risk factor for the development of adriamycin cardiomyopathy.

Echocardiography was found to be a useful, noninvasive technique for monitoring cardiac function in 62% of adult patients with cancer receiving adriamycin.

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Psychiatric Disorders

Demiurge*

William B. McGrath, M.D.

* Demiurge: A demonic being who attempts to collaborate with God in the creation of the world, spoiling it by introducing suffering and sorrow. (Plato, "Timaeus")

Human beings are more intelligent than insects and birds and animals. This is true. Yet they are wiser than we. They use their intelligence as it is meant to be used: to guide them to where their instincts can find propitious interchanges with the environment. Such is a fundamental principle of life.

We use our intelligence foolishly to try to distort our instincts or to deny them.

What long ago led us astray is the *delusion that mind controls our actions.* This is a delusion which is very, very hard to dispel. We cling to it in the face of terrible evidence that we are wrong. Unlike all other living creatures, we are at war with nature and with one another and with ourselves.

Witness a person, sitting in his room, brooding and introspective, trying to think his way out of his guilt and remorse. Maybe he will begin drinking or taking pills, wanting lethargy. Tomorrow he will go to a professional for analysis and antidepressants.

No! We want to shout at him: "Move, man! That is what all other creatures do." Only from them can he learn how to unshackle himself. They do not apologize or punish themselves for their God-given instincts. They obey God!

Outside that man's gloomy dwelling there is a world of abundance. His intelligence can serve no higher purpose than to guide him to places where he can play or mate or satisfy his curiosity or his need of beauty or awe.

The science of man ought to get back onto the highway of biology and stay there. This means identifying our own instincts as we do the wonderful instincts of insects and animals. As scientists we have to disengage ourselves from sociology and religion and metaphysics and from all the other non-science speculations.

There is no "power of mind over matter." Our actions are interchanges between our instincts and the situations in which they may or may not be gratified. Intelligence does not control! It can only teach us after the fact and thereby serve as a guide for the future.

Considering ourselves the center of the universe we refuse to acknowledge that we are an insignificant part of the whole of nature and creation. Trying to be demiurges, subordinate deities, we were banished from the garden of Eden. And until we learn the simple accepting humility of creatures we will not find our way back.

From: Dr. McGrath, Medical Director, Camelback Hospital, 5055 N. 34th St., Phoenix, AZ 85018.

Radiology

Case of the Month #31

Alan Frost, M.D.



Figure I: Upright



Figure II: Supine

An eleven-year-old Papago Indian female presented to the University of Arizona Hospital emergency room and was subsequently admitted. For approximately 48 hours prior to admission she had been experiencing anorexia, abdominal pain, and episodes of vomiting. Physical examination revealed decreased bowel sounds and diffuse abdominal guarding. Laboratory findings included hematocrit of 42, hemoglobin of 15.4, a normal urinalysis, and a white blood count of 14,300 with 64 polyps, 10 stabs, and 17 lymphs. What abdominal pathology as indicated by the radiograph and clinical history is responsible for the patient's problem?

From: University of Arizona Health Sciences Center, Dept. of Radiology, Dr. Frost, third year Resident.

Appendicitis

On the evening of admission the patient was taken to the operating room. Upon entering the peritoneal cavity, gross pus was immediately discovered. The appendix had to be freed from surrounding adhesions and was noted to be grossly gangrenous.

On the radiographs there is a narrow gas shadow extending in a vertical direction and overlying the right aspect of the sacrum. This is best visualized on the upright view and represents gas within the lumen of the appendix.

Intraluminal gas of the appendix may be seen normally if the appendix is situated in the right upper quadrant. This occurs when there is incomplete embryonic rotation of the midgut with the cecum remaining in the right upper quadrant. However, when gas is visualized within an appendix in its usual right lower quadrant location, it may be considered a reliable diagnostic sign of an acute appendicitis. In the acute abdomen this may be a difficult sign to discover as there is frequently overlying gas-filled small bowel.

This sign was first discovered by Musgrove in 1952 and has been seen by numerous radiologists since. Although one study of 25 cases of appendicitis had 9 with intraluminal appendical gas, other investigations show it to occur only rarely, perhaps in 2% or less of the cases of surgically proven appendicitis.

SUMMARY

Gas in the appendical lumen, although rarely seen, may be considered a reliable diagnostic sign of acute appendicitis when the cecum lies in the right lower quadrant.

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Special Articles

Medicine in Mexico

IX. Medical Education

Charles C. Daschbach

Nancy A. Chinnis

Professional health education in Mexico is currently entering a phase of development which questions the philosophy of medical and nursing education and ultimately where the responsibility should lie for quality, planning, and standardization. This era is not unlike the Flexnarian period experienced in American medical education at the turn of the century. It is the consequence of a proliferative anarchy in the establishment of new medical schools, overcrowded teaching conditions, and the lack of a national policy which will direct future planning.

During 1976 the Mexican Ministry of Public Education tried to survey the 55 medical schools in the Republic. Four of these had just been founded and of the remaining 51, 38 responded cooperatively to the survey, revealing an extremely serious situation.[1] Interestingly, 30% had been founded within the last 6 years. First year enrollments in 1970 were 8,650 and had risen to 20,001 in 1975, while the total number of medical students had increased from 31,152 to 67,308 for the same period. The desertion rate averaged 39%, varying from 12% to 81%, and the average student-teacher ratio was 9:1 with a range from 4:1 to 38:1. Over 7,000 medical students graduated in 1976 and the government projection for 1979 is for over 12,500.

While there is a diverse spectrum of individual medical schools, the National

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Autonomous University of Mexico (UNAM) in Mexico City serves as the prototype for Mexican educational institutions. One third of all Mexican medical students attend the UNAM School of Medicine, with a total enrollment of 24,000 medical students in 1976. The UNAM teaching faculty employs 3,561 instructors, about 45% of all Mexican medical school professors, giving a student teacher ratio of about 6 to 1. The desertion rate averaged over 30% for 1970-1975 while total enrollment increased by almost 80%. Following the Hispanic tradition of university autonomy, the federal government has little control over the institution, while ironically supplying most of its budget.

The incomplete data collected in the 1976 survey reflects a highly complex situation which has developed rapidly and will be difficult to correct. The tremendous number of aspirants to the medical profession is not unfamiliar even to developed nations, but it is aggravated by current socioeconomic factors in Latin America. The policy of "limited admissions" with respect to teaching capacity is not practiced, resulting in greatly strained limitations of physical resources. The high incidence of desertion expresses among many things an irregular quality of students, incomplete motivation, and general academic deficiencies.

In contrast to the medical schools, the nation's 110 general nursing schools graduated only 2300 students in 1975, and are not significantly increasing their enrollments. Only ten schools offer specialized training for nurses, and there are over 140 centers for nursing auxiliaries and technical nurses, which vary spectacularly in the type and quality of education offered. This disequilibrium in the small number of nurses to doctors is accentuated by an urban hospital system patterned after the American-European style of division of hospital labor.

The Secretary of Health and Welfare has begun to assume a degree of responsibility for the quality of medical education in Mexico. This ministry recently instituted a new "professional examination" which must be passed by all graduates. On the other side of the issue, defendants of the rapidly growing medical education system point to Mexico's increasing population and to the current physician to patient ratios in rural areas. While the call has been made for a national policy to direct the adjustments needed in Mexican health education, the future is uncertain. The ensuing national debate will involve not only the issue of quality education, but also the right to that education and the quality of life itself.

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The Internal Medicine Clinical Nurse Specialist

Cindy Lou Kohn, B.S.N.

Health care institutions are being stressed by increasing public demand for more and better care. One solution to providing the demanded services is extend the role of the nurse as a provider of health care. Settings for utilization of nurses as care providers include hospital out-patient departments, health maintenance organizations, rural health clinics, private physician offices and independent practice.

Many different terms are being used to label nurses in the extended roles, with the most common being nurse practitioner, nurse clinician and clinical nurse specialist. Nurse practitioners are registered nurses who have completed an approved program in the assessment of the physical and psychosocial health status of individuals, families or groups in a variety of settings. Licensure or certification of nurse practitioners are offered by certain states. The nurse clinician demonstrates expertise in nursing practice and is capable of implementing both direct and indirect care. Minimal preparation for this role is the baccalaureate degree. Clinical nurse specialists have graduate preparation in nursing preferably with an emphasis in a specialized area of clinical nursing. This person primarily functions in the hospital setting.

The purpose of this paper is to describe the role of a clinical nurse specialist in a general internal medical clinic.

The internal medicine clinical nurse specialist at the University of Arizona Health Sciences Center is academically prepared at the master's degree level in nursing. Along with didatic courses the nurse has had a ten month practicum in both adult ambulatory care settings and emergency departments under the supervision of physicians.

As a clinical nurse specialist in a general internal medical clinic the nurse's primary responsibility is to provide comprehensive patient care by accepting certain responsibilities formerly assumed by the physician. Specific duties include obtaining patient histories, performing physical examinations and ordering and interpreting laboratory tests in order to identify the health status of the individual. The nurse provides on-going management of the patient and schedules follow-up appointments as needed.

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The clinical nurse specialist is responsible to three physicians in the department of internal medicine who are consulted in the medical clinic for interpreting the findings on physical examination and diagnostic work-up and planning the treatment regimen. All entries in the medical record are countersigned by the attending physician. Prescriptions are signed by the attending physician and permission of the physician is required to change medications.

The unique setting at University Hospital lends itself to collaborative patient care, providing continuous exchange between the clinical nurse specialist, physicians and other members of the health care team. When patients present with complex problems there is always the availability of consultation with specialists.

For the purposes of this paper four and a half months of practice (five half days per week) was analyzed; the clinical nurse specialist was caring for 120 patients ranging in age from 16 to 86 years. The patients seen by the nurse were selected a random from patients calling in for appointments in the general internal medical clinic, regardless of the severity of their illnesses. Table I shows the problems represented by these patients.

The variety of medical problems ranged from routine health care screening to diag-

TABLE I

Distribution of Problems	Number of Patients
Hypertension	30
Gastrointestinal	22
Health Maintenance	20
Musculoskeletal	19
Metabolic	19
Cardiovascular	16
Psychiatric	14
Dermatologic	14
Gynecologic	13
Neurologic	10
Infections	10
Genitourinary	10
Hematologic	9
Neoplastic	8
Respiratory	7
Endocrine	7

N.B. Total of problems is greater than the total of patients since most patients had more than one problem.

noses requiring major surgical treatment. For example, the category of neurologic problems included such diagnoses as migraine and tension headache, dizziness, vertigo of uncertain etiology and brain tumor. Patients with ischemic heart disease, including angina, post-myocardial infarction and post-coronary artery bypass were listed under cardiovascular problems. Neoplastic disease varied from cervical dysplasia to adenocarcinoma of the kidney.

The clinical nurse specialist has other responsibilities associated with the position such as providing consultation in areas of special interest: management of chronic diseases, primarily hypertension and diabetes, gynecologic problems, patient education and psychosocial aspects of care including short term counseling. Since the clinical nurse specialist is able to provide continuity of care and spend more time with patients, the nurse develops a comfortable working relationship promoting expression of psychosocial problems.

Extension of clinic services is provided by the clinical nurse specialist by monitoring general medical clinic patients admitted to the hospital. The nurse is available to hospital staff for patient conferences and discharge planning coordinating in-patient and out-patient services. The clinical nurse specialist participates in a home follow-up program for one or two visits after discharge to those patients in need of education regarding self care or family instruction. After two home visits the clinical nurse specialist provides referral to other agencies as necessary.

The clinical nurse specialist functioning in a university setting is in an ideal position to do research and is encouraged to make contributions to the nursing profession with documentation and publications of ongoing clinical research. In addition to original research, the nurse attends monthly research seminars to remain informed of recent advances in general medicine.

It is expected that the clinical nurse specialist will keep informed and current in the development of knowledge of general medicine. In order to enhance professional growth, the nurse participates in nursing and medical self-education, attends teaching conferences, seminars and grand rounds and participates in patient care conferences.

At Arizona Health Sciences Center, the clinical nurse specialist in internal medicine serves a key function in the provisions of comprehensive patient care and is capable of handling the majority of patient problems seen in an out-patient general medical clinic. As described above, the clinical nurse specialist assumes an important and unique role in promoting and maintaining health.



"Not invisible, but unnoticed, Watson. You did not know where to look, and so you missed all that was important."
--Sherlock Holmes

Jane M. Orient, M.D.

Since 1955, when Zollinger and Ellison reported three cases of virulent and atypical peptic ulcers associated with islet cell tumors of the pancreas, the rate of recognition of such cases has accelerated. There are now over 1000 cases in the literature, and of course many more unreported. The frequency of the syndrome among all patients with peptic ulcer is unknown; one author guessed about 1%. Still it has stimulated many reviews, since from a scientific standpoint it represents one of those experiments of nature whose pathophysiology can be rather clearly understood and thus lend insight into the more common yet more heterogeneous and subtle duodenal ulcer. From a clinical standpoint, to miss the diagnosis is a disaster, for the most routine appearing duodenal ulcer can develop severe complications within hours of the violation of the gastrointestinal tract by standard surgical procedures.

The Greeks originated the theory of disease that was predominant throughout the Middle Ages. They believed the body to be composed of four humors: blood, phlegm, yellow bile, and black bile. Disease resulted from an imbalance in the humors, and was corrected by applying an opposing element. Today we have discovered many more humors. Within the gastrointestinal tract alone, around a dozen polypeptides have been identified as possible hormones. One of the first discovered and best understood is gastrin. Its main source is normally the antral mucosa. Its secretion is stimulated by vagal impulses, protein, gastric distention, and (at least per-

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The Recognition and Management of Zollinger-Ellison Syndrome

missively) by calcium. Its release is inhibited by secretin, glucagon, and most importantly, by acid.

The Zollinger-Ellison syndrome is the hypersecretion of gastric acid in response to an excess of gastrin, produced by a tumor (a gastrinoma) despite the presence of its negative feedback inhibitor. An algorithm for diagnosis and management is presented as a guide; some points are controversial. The limits stated for normal and unequivocally elevated gastrin levels will vary somewhat with the laboratory.

The clinical presentation of Z-E may not arouse suspicion. Until 1970, it was an aggressive disease as seen at the Mayo Clinic, in accord with its reputation. But of 40 patients seen since that time, 19 had only the usual symptoms of peptic ulcer disease. Eleven had diarrhea in addition to the abdominal pain, one had diarrhea only, and nine had ulcer complaints plus some feature of multiple endocrine adenomatosis. None of the patients had ulcers in atypical locations, and many had only erosive duodenitis at endoscopy. Thus it is necessary to obtain a serum gastrin level on any patient for whom surgical treatment is contemplated.[1]

There are several clinical features which should raise the index of suspicion, through their absence is not reassuring:

1. Very young or very old patient.
2. Perforation or hemorrhage occurring post-partum or soon after a standard ulcer operation.
3. Severe diarrhea, often accompanied by steatorrhea, and relieved by gastric suction.
4. Atypical location of ulcers, e.g. distal duodenum, or jejunum.
5. Family history of ulcer disease, endocrine disease, and/or nephrolithiasis, or personal history of same.
6. Radiologic findings: fluid retention in stomach, hypertrophy of gastric mucosa, intestinal hyperperistalsis, duodenal dilatation, or blunted valvulae conniventes.
7. Excessive gastric acid:
 a. Basal output of >15mEq/hr (present in up to 10% of patients with ordinary ulcer, and absent in 30% of Z-E cases.)
 b. Basal/maximum output ratio of >0.6 (present in about 5% of controls and about 50% of Z-E patients.)
 c. Output of >5mEq/hr after an acid-lowering operation.[2]

The only reliable diagnostic test is the serum gastrin level. If the value is not high enough to be diagnostic, a provocative test may be used. The best is the infusion of secretin, which normally should suppress gastrin, but paradoxically raises it in Z-E, in about 75% of cases. A calcium infusion normally will raise gastrin level, but only slightly. An increase of >490 pg/ml is diagnostic for Z-E.[3]

If the serum gastrin is elevated, gastric acid secretion must be measured, since over-production is to be expected if the normal inhibitor is lacking. This occurs in pernicious anemia, truncal vagotomy, and chronic atrophic gastritis. Gastrins may be in the Z-E range frequently.

The combination of hypergastrinemia and hyperacidity can occur with chronic renal failure, acute gastric outlet obstruction, or short bowel syndrome. In a patient who has had gastric resection, isolated retained antrum must be ruled out. If possible, the slides of the duodenal margin

of resection should be reviewed for the presence of Brunner's glands. The antral cells still respond to normal stimuli; they are not bathed by acid from the stomach, but they can be inhibited by an infusion of secretin.[4]

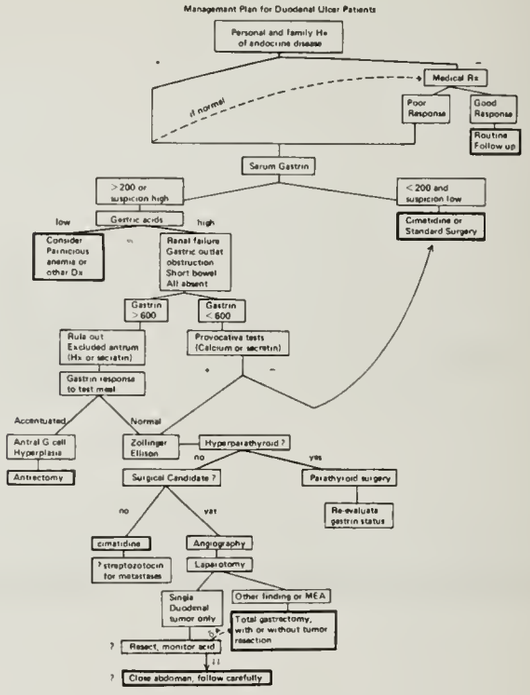
In some cases of severe hypergastrinemia and hypersecretion, no tumor has been found, but the gastrin-secreting cells (G-cells) of the antrum have been hyperplastic on immunofluorescence staining. The history tends to be short. Several patients have been cured by antrectomy. Some have proposed that this be called Z-E Type I[5]; the consensus is that the term Z-E should be reserved for cases of gastrinoma, however. It is not clear how G-cell hyperplasia differs, other than in degree, from ordinary duodenal ulcer. It is rare, being found in only 3 of 400 patients with peptic ulcer and hypergastrinemia or recurrent ulcers post surgery.[6] It is distinguished from Z-E by the gastrin response to a meal. Patients with G-cell hyperplasia (and many duodenal ulcer patients) show an exaggerated serum gastrin response to protein, in contrast to Z-E patients.[4]

Having excluded the entities above, the patient with hyperacidity and either sufficiently high gastrin level or a positive provocative test is diagnosed as having Z-E. The prognosis and therapy depends on the location of the tumor, its malignancy, and the associated findings.

The source of the gastrin may be hyperplasia of the pancreatic islets, and/or one or more islet cell adenomata located either in the pancreas, duodenum, or rarely elsewhere. Malignancy is not possible to ascertain histologically, but is defined by the behavior of the tumor. About 60% are multifocal or metastatic at the time of diagnosis. The odds are better with tumors in the duodenum; 47% of them are single[2] in contrast to about 25% of the pancreatic lesions. Tumors may be found pre-operatively by angiography in 30-50% of patients. At laparotomy they must be searched for in liver, regional lymph nodes, pancreas, and duodenum. The last should be mobilized and opened for better palpation, and the pancreas biopsied for hyperplasia.

About half the patients with Z-E will also have the syndrome of multiple endocrine adenomatosis Type I (MEA I), Wermer's syndrome. Conversely, peptic ulcer disease is the most common mode of presentation of MEA. This is inherited as an autosomal dominant trait. The clinical manifestations are variable, and tend to increase with age. The frequency of occurrence of various tumors in MEA I is: pituitary adenoma (65%); islet cell adenoma or carcinoma (81%); parathyroid adenoma or hyperplasia (88%). Less commonly associated are: Adrenal cortical adenomas (38%); thyroid adenomas (19%); carcinoid tumors; lipomas, usually multiple; and mediastinal endocrine neoplasm. The islet cell neoplasms most commonly manifest themselves as hypoglycemia (insulinoma) or ulcer diathesis (gastrinoma).[8]

To understand the occurrence of these adenomata as a complex, and the fact that the humors they secrete may be multiple and often "ectopic," the concept of the APUD system is helpful. The system is named for one of the



characteristics of the cells, *A*mine *P*recursor *U*ptake and *D*ecarboxylation. Early in embryogenesis, they migrate from the neuroectoderm (probably the neural crest) into endocrine glands such as the pancreatic islets, the pituitary, the adrenal medulla, and the thyroid (forming the calcitonin-producing cells); or into the gut as enterochromaffin cells, a diffusely distributed endocrine system. Parathyroid cells have not been shown to be APUD cells, but are thought to have a neuroectodermal orgin. The MEA syndromes are then regarded as forms of neuroectodermal dysplasia.[9,10,11]

Several mechanisms have been proposed to account for the tumor formation. One postulates a pituitary factor. The evidence for this is scanty, and consists mainly in the effect of growth hormone on gastric mucosa.[5] Vance has theorized that nesidioblastosis, or hyperplasia of the pancreatic islets, is the primary defect. A suggested sequence of events would be: glucagon excess (or gastrin)— > release of calcitonin and other effects— > decrease in serum calcium— > parathyroid hyperplasia— > tertiary hyperparathyroidism.[12] Wermer thought that all the cells had a hereditary defect which manifested in any that had a factor required for response. Knudson's mutation model for tumor formation states that tumors result from mutations occurring in each of two homologous genes. In the hereditary syndromes, one of the mutations is already present in all cells. The occurrence of the second is a relatively likely event, compared with both arising de novo, so that tumors tend to be multifocal. Examples are the hereditary form of retinoblastoma, familial polyposis, and familial pheochromocytoma (bilateral in 50-80% compared with 6% of sporadic cases).

An understanding of MEA brings to mind: 1) the need to screen relatives of Z-E patients; 2) the need to consider Z-E in patients with a suggestive family history; 3) the

possible interrelationships between APUD tumors; 4) the likelihood that tumors will be multifocal.

The most common interrelationship is that between gastrinomas and hyperparathyroidism. Though peptic ulcer has been said to be doubled in incidence among patients with primary hyperparathyroidism, later series have not found this to be so. Probably any increase represents unrecognized cases of MEA. Since hypercalcemia does augment gastrin secretion, however, it may make the clinical picture of Z-E much more severe. Also, in patients without MEA, parathyroidectomy has resulted in lowered gastrin values and improved symptoms. Thus, in a patient with Z-E who is hyperparathyroid, it is reasonable to operate on the parathyroids first, circumstances permitting.[13]

Surgical therapy for Z-E is usually a total gastrectomy. Hypergastrinemia itself has no serious physiological consequence in the absence of the end organ. Most deaths in Z-E result from peptic ulcer disease. Though frequently metastatic, the tumors tend to be indolent. Metastases are very often too small for detection. It is worthwhile to resect pancreatic adenomas which appear to be single, in conjunction with the total gastrectomy. Resection of the duodenum and pancreas as a curative "cancer operation" is not worth the serious morbidity and mortality; in combination with total gastrectomy it is hardly compatible with life. Of 265 patients listed in the Z-E Tumor Registry, who were treated with total gastrectomy, 30% have died, 60% of these due to post-operative complications. Patients who survive the surgery do surprisingly well. In contrast, of patients who undergo lesser procedures, 49% are dead within one year, 72% within 10 years. Much of the surgical mortality is due to the severe nature of the disease; performed early and electively, it may be as low as 5%.

An exception to the general rule about total gastrectomy may be patients in whom a single duodenal carcinoid is found. In about 50% of cases, a local tumor excision suffices. In the remainder, a total gastrectomy eventually becomes necessary.[2,14,15,16] Gastric acids should be monitored at time of surgery and after, as they may decrease dramatically within hours, if total tumor excision is accomplished. On the other hand, disease may recur years later.[17] Probably no lesser operation on the stomach should be done. Tumors in MEA should be assumed to be multifocal; virtually all (92%) in Ballard's series were.[8] The severity of the disease may make a simple tumor excision unfeasible. It is important to realize that a duodenal carcinoid may be non-functional, an incidental finding in ordinary peptic ulcer disease,[16] as in the following case seen at Parkland Memorial Hospital by the author.

Case Report. G.K. was a 55-year-old white man who complained of 3 weeks of vomiting, diarrhea, and weight loss. A pre-pyloric ulcer with gastric outlet obstruction was seen. Basal/maximum gastric acids were 5.7/51 mEq/hr, the gastrin level was 45, and the serum calcium 8.9. He underwent antrectomy with Billroth II anastomosis. Pathology showed nests of cells in the duodenum, read as pancreatic islet-cell carcinoid. His course was complicated by a duodenal-cutaneous fistula, and hemorrhage. At endoscopy, a suture was removed from the site of a pre-anastomotic ulcer. Post-op acid secretion was 0.5 mEq/hr (basal). He recovered well, and a laparotomy a year later for trauma showed no evidence of tumor.

Since the Histamine H_2 receptor antagonist cimetidine became available, patients have been successfully treated with medical means alone.[17] The long term effects are as yet unknown, however. The most serious side effect yet reported has been the perforation of chronic ulcers within 2 weeks of stopping therapy, in patients not suspected of having Z-E.[18] Furthermore, the occasional discovery of an isolated tumor probably makes an exploratory laparotomy worthwhile in most cases.

In summary, even that special case of peptic ulcer disease, the Z-E syndrome, has been found to be heterogeneous. The appropriate treatment hinges critically upon a precise diagnosis.

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Controversies In Medicine

Indications For Coronary Artery Bypass Surgery

The great amount of heat generated by the coronary artery bypass operation has no doubt confused many physicians regarding the indications for the operation.

A cardiologist, Dr. Desser, and a cardiovascular surgeon, Dr. Diethrich, perceive the problem slightly differently but both share a common belief, that these patients should have the best available care. Their thoughtful positions highlight some of the uncertainties which still remain in this area. MBB

Kenneth B. Desser, M.D.

A Cardiologist's View

Edward B. Diethrich, M.D.

A Cardiovascular Surgeon's View

The advent of coronary bypass surgery has evoked an extraordinary amount of controversy in the medical community. Indeed, the salubrious benefits of pharmacologic therapy vis a vis treatment of patients with chest pain by means of scalpel appears on a monthly basis in the contemporary literature. The cardiovascular body politic is basically comprised of three groups, to wit: 1) those who espouse the thesis that major coronary artery disease is an absolute indication for bypass surgery, 2) the skeptics who aver that such dramatic intervention is merely yet another therapeutic misadventure which will surely merit a minor footnote in the history of applied biology and 3) those who posit a limited but utilitarian role for surgery in selected cases. Interestingly, all three groups can adduce scientific papers to support their respective views.

The purpose of this article is to provide a tempered overview of the medical indications for coronary bypass surgery.

Absolute Indications include angina pectoris secondary to bypassable left main coronary artery disease[1] and recurrent debilitating anginal attacks which are considered refractory to optimal pharmacologic interventions. Such drug therapy should be rendered by physicians who are well versed and skilled in the application of nitrites, beta blocking agents, ancillary medication and exercise programs.[2] When medical therapy does not afford clinical improvement, relief from pain has been accomplished by surgery in the vast majority of operated subjects with adequate left ventricular performance, reversal of stress-induced ischemic electrocardiographic patterns, and increased work tolerance as gauges for evaluation.

From: The Institute for Cardiovascular Diseases, Good Samaritan Hospital, 1033 East McDowell Road, Phoenix, AZ 85006. Supported in Part by the E. Nichols and Kim Sigsworth Memorial Funds, and The Institute for Cardiovascular Diseases, Inc. Address for Reprints: Kenneth B. Desser, M.D., Good Samaritan Hospital, P.O. Box 2989, Phoenix, AZ 85062.

Throughout the history of medicine, controversy regarding the medical versus surgical treatment of disease entities has run rampant. The current dispute[1] involving the exact role of the coronary artery bypass (CAB) operation in the treatment of atherosclerotic occlusive disease of the coronary arteries is no exception.

When the CAB procedure was first introduced,[2] skeptics were convinced that a direct bypass from the aorta around a coronary arterial obstruction would do little more than mimic the positive psychological effects witnessed in some other operations, like internal mammary artery ligation.[3] It was not long, however, before objective scientific studies were demonstrating a level of relief of myocardial ischemia via the bypass operation well beyond that which could be considered emotionally related.[4,5,6] The presence or absence of angina pectoris as the major determinant of myocardial ischemia now became less significant a parameter of cardiac function. In its place came enhanced left ventricular performance, reversal of stress-induced ischemic electrocardiographic patterns, and increased work tolerance as gauges for evaluation.

In general, the selection of patients for myocardial revascularization is based upon the specific location of the coronary arterial narrowing and the extent of potential myocardial injury should the blood flow be further reduced. Also to be considered are any pulmonary, renal, hepatic, or metabolic diseases present. Complete assessment of the left ventricle and associated cardiac and vascular lesions must be made. Interestingly enough, age is no longer an important factor in determining operability.

From: The Section of Cardiovascular Surgery, Arizona Heart Institute, Phoenix, Arizona. Reprint requests to: Coordinator, Academic Affairs, Arizona Heart Institute, P.O. Box 10,000, Phoenix, AZ 85064.

Dr. Desser

cular function. Those patients afflicted with main left coronary disease fare better in terms of survival after bypass.

Relative Indications are construed by definition to be intelligible only in relation to something else or qualified by an antecedent. "Unstable" angina pectoris is considered to be such an indication. Now the definition of "unstable" is subject to the vagaries of those respective investigators who enunciate an advocacy for either medical or surgical intervention and all such criteria must be viewed with circumspect.[3] Greater symptomatic improvement has been demonstrated in those cohorts who undergo coronary artery bypass.[4] If a patient initially responds to medical therapy for unstable angina pectoris and subsequently expires, the attending physician need not suffer under the yoke of opprobrium since the two year survival rate in the medically and surgically groups is not significantly different.[1] It is no surprise that a profound degree of equivocation has been evinced by cardiovascular pundits in discussions of the "intermediate syndrome" or "coronary insufficiency" since the criteria for medical vs surgical therapy are in a state of flux.

Possible Indications are inherently unproven and incite polemics of high order. Stable angina pectoris arising as a result of angiographically demonstrable double or triple vessel coronary disease with major obstruction of the proximal left anterior coronary artery portends susceptibility to major myocardial infarction or demise. Physicians who do not undertake the care of such cases could reasonably request a compilation of data on the subject and be narcotized into a state of bemused diffidence. The fact that surgical therapy in these patients, exclusive of main left coronary disease, does not improve three year longevity[5] in no way vitiates the salutary influence of the procedure on symptoms. All current randomized prospective investigations do not support the precept that bypass surgery enhances the "quantity" of life in this setting. "Quality", however, is a praiseworthy goal which should be considered in relation to the patient's lifestyle. An 80 to 90% chance of being untethered from a life of repose, replete with the ingestion of 10 to 20 pills per day has been proposed, a fortiori, as a compelling reason for surgery which has a 5 per cent or less mortality.

Unproven or Experimental Indications are exemplified by: 1) Bypass procedures during the acute stage of myocardial infarction. 2) Surgical intervention in an asymptomatic patient with significant coronary artery disease demonstrated by angiography. The latter is usually performed because of a positive exercise stress test, suggestive physical findings or an abnormal resting electrocardiogram. 3) "Prophylactic" revascularization in those with bypassable coronary disease who are angina-free following a myocardial infarction. 4) Graft implantation (exclusive of aneurysmectomy) for intractable ventricular tachyarrhythmias, 5) Operation for "variant" angina pectoris shown to be based on coronary artery spasm over an atherosclerotic substrate. 6) Potential reversal of

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Patients with coronary artery disease can be grouped according to the severity of arterial involvement and symptomatology. In the *high-risk category* are patients with greater than fifty percent stenosis of the left main coronary artery[7] and equivalent narrowing in the circumflex and anterior descending arteries. Operative intervention is strongly indicated even for the asymptomatic patient, since the potential for sudden death is greatly increased with this lesion pattern.

With the exception of patients with the life-threatening lesions noted previously, the majority of symptomatic patients may be grouped together. In this *mid-risk group*, the advisability of operative treatment is related to the severity of the symptoms, the patient's incapacitation, and the success of medical management. From an arteriographic standpoint, patients in this category exhibit double or triple vessel disease, with at least one lesion producing seventy-five percent or greater narrowing in the proximal segment of a major coronary artery. A typical example of arterial involvement might be: ninety percent occlusion of the right, sixty percent of the anterior descending, and fifty percent of the obtuse marginal branch of the circumflex artery. Even some patients with single vessel disease may be classified with this second group, namely, if the lesion involves the proximal anterior descending coronary artery. Programs of exercise, weight reduction, blood pressure control, dietary restriction, and medication (vasodilators and beta adrenergic blocking agents) should be instituted initially; however, if symptoms persist, myocardial revascularization should be recommended.

The last grouping of coronary arterial disease patients is generally reserved for single (or multiple) vessel disease of a *subcritical nature*. As with the second category, most of these people may be treated medically with success. On the other hand, some patients' symptoms may be uncontrollable medically, in which case operation may be advised. Others may demonstrate an intolerance to medications, thereby making an operation necessary for relief of symptoms. Also in this group would be found post myocardial infarction patients with additional coronary lesions. Statistically, these people have a significantly increased risk of future infarctions and death.

My experience with the coronary artery bypass procedure has shown it to be of significant benefit in the treatment of myocardial ischemia. In patients with severe disease, such as left main lesions or multiple vessel involvement, the operation has definitely prolonged life. By revascularizing the myocardium, it has been possible to prevent myocardial infarction in patients with unstable angina and high-risk lesions. Further, anginal symptoms have been relieved, and many cases of stress-induced, preoperative ischemic changes have been reversed. In conjunction with this, the quality of life for many has been enhanced significantly. However, the basic disease process is unaltered. Future research efforts must be directed toward detecting, reversing, and preventing atherosclerotic disease.

Dr. Desser

ventricular asynergy in myocardial segments with borderline perfusion.

Despite the frequent stentorian exclamations and authoritative visage of the dramatis personae, including both physicians and patients, case reports extolling the virtues of such exercises merely represent anecdotal accounts and should be viewed with caution. These seemingly astonishing feats are reminiscent of magazine advertisements for panaceas which are thought to "cure" obesity, i.e. before therapy a picture of a grim, depressed 300 pound human being, 6 weeks after therapy a 150 pound person of felicitous countenance. Problem: how many try, how many fail and where is the 2 year photograph?

Absolute Contraindications for bypass surgery include angina pectoris consequent to coronary spasm in otherwise normal arteries and atherosclerotic cardiomyopathy with congestive heart failure as the *dominant* clinical feature. The vast majority of studies have provided irrefutable evidence that bypass surgery is not beneficial in those patients with severe myocardial disease and pump failure. Additionally, the operative mortality is prohibitive when compared with the results. One important caveat: an occasional patient has been observed to improve symptomatically after revascularization for a low output state. Invariably, such subjects have also been placed on new medical regimens including large doses of pre-load and after-load reducing agents which are currently considered experimental. These considerations provide indirect evidence that the coronary bypass procedure per se is not the sole factor influencing the clinical result.

The above comments have been directed to isolated bypass surgery and it should be kept in mind that revascularization is frequently applied in combination with other cardiovascular procedures. When originally conceived, bypass surgery appeared to be another unorthodox therapeutic modality. It is abundantly clear that the operation, when utilized in a selective fashion, is firmly established as a major form of therapy for coronary disease. The evolution and velocity of investigative data pertinant to such techniques is accelerating at a rate which may relegate this manuscript to the archeological shelves before its publication.

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OUT OF CONTEXT (DRY GULCH)

John W. Kennedy, M.D.

Digitalis Revisited

For some who traversed the medical school ramps four decades ago, Digitalis remains one of the few prescribed drugs that are recognizable from that era. A recent review by Dr. E.S. Brady of the history, chemistry, pharmacokinetics, and therapeutic use of this, now somewhat ancient but necessary and useful drug, he concludes in the following fashion. "Digitalis remains an enigma, in part, to the physician, pharmacologist, and plant chemist. It is not wholly predictable in its effects on the patient, its mode of action has not been clearly defined, its glycosides defy synthesis, and its clinical assays pose problems. Perhaps its blossoms were intended only to provide gloves for the fairy folk or caps for trolls, and it is this magic that gives strength to the failing heart. While the poet may be content with this explanation, the medical scientist has already begun the third century of his search to solve the therapeutic riddles of this common foxglove."

Digitalis Reharvested, Edward S. Brady, M.D. Drug Therapy, Oct. 1977, Pg. 71.

"Excellent Versus Adequate Care" That Is the Question!

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With the increasing sophistication of medical diagnosis and treatment, the costs for medical care in the United States is tremendous. Furthermore, with emphasis on preventive medicine the cost for taking care of healthy patients is rivaling that of the outpatient care of the ill. It is true that we all want our population to have the best medical care available. However, the cost of this approach is occupying an ever-increasing percentage of our gross national product. This has resulted in plans to contain its growth. The ways that have been suggested involve increasing efficiency and/or increasing competition and/or rationing the available resources.

The problem with increased efficiency is that we, as physicians, and hospitals, as administrators, are concerned about efficiency as it relates to our own domain but not as it relates to the general community in which we find ourselves. Increasing our own efficiency leads to greater rewards but does not necessarily lead to overall cost savings for the nation. The recent controversy regarding CAT scanners is a typical example, but similar stories can be told for EEG machines, thyroid scanners as well as centers that do cardiovascular diagnostic and surgical procedures. These machines and procedures do wonders for their owners but lead to much duplication of spending which is detrimental to the cost of our health system. Centralizing medical care within one or two institutions and decentralizing it elsewhere has until this time not proved successful in the private sector for obvious reasons. However, such centralization has taken place in the Uniformed Services Health System and this is a model which one could point to to demonstrate that such a process could occur and function. The Central Arizona Health Systems Agency has been set up to perform such a function for us within our present health care delivery system. But as we all know, the agency has not been uniformly accepted nor has its decisions been received with acceptance from all sectors.

Increasing competition would theoretically produce a more efficient operation with lowering of costs. However, it has become apparent that physicians as suppliers also determine the demand for their services and thus overpopulating a region with more physicians than are needed just results in greater utilization of the available services.

The last option, that of rationing medical care, is probably the most difficult to deal with for the practicing physician. How can a physician who wants to do the best that he can for a patient make the decision to withhold certain therapy because the patient does not fulfill certain criteria previously established for that treatment? These ethical questions have arisen in the past as new therapies have been introduced. For instance, chronic renal dialysis initially was started on a limited scale and since there was an over supply of patients, ethical decisions had to be made concerning which patients would be treated. Patients with diabetic nephropathy who had retinopathy and peripheral vascular disease were initially excluded from such programs. However, once Medicare underwrote the treatment of chronic dialysis these same patients were now readily accepted into the markedly expanded programs that were available coast-to-coast. Similarly, when coronary care units were first introduced certain criteria were established for admission. As these beds increased in number the criteria for admission slowly were changed to allow greater utilization. Thus, in order to ration medical care difficult questions and answers are going to have to be made. If rationing of medical care is to work we will be moving from a system in which everyone tried for excellence, to a system where adequate medical care is going to be administered to all. The term "adequate" will no doubt reflect in certain instances excellent medical care and in other instances some value judgments regarding our supply and demand capabilities in reference to our overall national economy.

It would appear that of the three alternatives the last is probably the one which will be utilized to contain costs. This will be accomplished by setting up criteria for utilization (utilization review committees and PSRO's), criteria for diagnosis and treatment (medical in- and out-patient audits mandated by the JCAH) and

centralization of medical care and equipment (CASHA). In addition, a regional fee schedule will be developed based on existing fees in the community. If one looks at what is developing one can't help but feel that we are in a chess game and are being checkmated subtly but surely. What is sad, is that we could have done by ourselves all that is now being done from above, if we only weren't so short-sighted several years ago.

Marshall B. Block, M.D.
Editor

Research in Anesthesiology

The Department of Anesthesiology, the newest of the University of Arizona College of Medicine's fifteen Departments, is conducting basic and clinical research with implications not only restricted to the discipline of anesthesia, but of wider scope. A summary of some of the investigational projects of this Department follows.

1. *New anesthesia and respiratory gas sensors.* The present methods of analyzing respiratory gases and anesthetic vapors, whether in the pulmonary function laboratory, at the bedside, or in the operating room, entail laborious, expensive, and difficult to calibrate analytic techniques such as mass spectroscopy, infrared analysis, and gas chromatography. Lawrence Adler, M.D., and Jerry Calkins, M.D., Ph.D., of the Department of Anesthesiology and Warren Jewett, Ph.D., Research Associate in Anesthesiology and in the College of Engineering, have tackled this problem from two approaches. In one, a sensor has been developed in which a silastic band loses elasticity proportional to anesthetic concentrations. A prototype of this device has been used successfully during anesthesia at University Hospital. The other promising approach with a broad range of applications has been the development of a sensor responsive to all gases and vapors based on a fluidic principle. In this case a mixture of gases can be discriminated and accurate concentrations determined. This rugged, economical gas analyzer has applicability not only in the operating room, but wherever respiratory gas analysis is required. Prototypes will soon be evaluated in various clinical situations. From an anesthesia standpoint, both sensors will allow better minute-to-minute control by the anesthesiologist and also enable widespread use of so-called "closed circuit" anesthesia techniques. This results in economy of expensive anesthetics and prevents spill-over of these vapors into the O.R. as environmental pollutants.

2. *Viscerotoxicity of halogenated anesthetics.* The Department has developed and published investigations concerned with development of animal models simulating the entity of so-called "halothane hepatitis". Research by Burnell R. Brown, Jr., M.D., Ph.D., and I. Glenn Sipes, Ph.D., continues to evaluate hepatotoxicity of anesthetics based on abnormal biotransformation routes. A promising note is that the compound diethyldithiocarbonate (DDC) can prevent this toxicity in animals. After further animal studies, it is hoped to evaluate DDC clinically in this rare but frequently lethal toxicity. Studies are underway to establish methods of diagnosis for this condition which so closely mimics viral hepatitis. Casey Blitt, M.D., has recently been awarded a National Institutes of Health research grant to study pulmonary biotransformation of inhalation anesthetics, an area which has not been explored before. He is pursuing investigations to ascertain if biotransformation plays a role in development of atelectasis, perhaps the most common complication of surgery and anesthesia.

3. *Carcinogenicity of anesthetics.* Robert Vaughan, M.D., Jay Gandolfi, Ph.D., and Dr. Sipes are actively pursuing laboratory studies to determine whether clinically employed inhalation anesthetics are potentially carcinogenic. The structural similarity between halogenated anesthetics and known carcinogens such as divinyl chloride and trichloroethylene is striking. This is an area of considerable public health concern since 20 million patients and millions of O.R. personnel are exposed annually to anesthetics, both in pharmacologic and trace dosages.

4. *Clinical studies.* Stuart Hameroff, M.D., and Dr. Brown are continuing investigations in the Pain Clinic of the drug cyclobenzaprine (Flexeril, MSD) developed in part at the University of Arizona. Dr. Blitt is assessing the effects of the pre-operative medication droperidol on the incidence of induction cardiac arrhythmias. Charles Otto, M.D., is performing investigational studies of respiratory mechanics in severely ill patients in the Intensive Care Unit.

The research of the Department of Anesthesiology is vigorous and overlaps many areas in the broad field of medicine. The questions being studied have important practical value to the physicians of Arizona.

Burnell R. Brown, Jr., M.D., Ph.D.
Department Head
Anesthesiology

Louis J. Kettel, M.D., Dean
College of Medicine

Auxiliary Highlights

Since long before recycling became fashionable, Medical Auxiliary has enjoyed the return and rekindled interest of active members who may recess their activity from time to time for other volunteer emphasis. When they return to the Auxiliary scene, they bring new dimensions acquired from a varied assortment of additional interests.

Mrs. James L. Parsons, installed as State Auxiliary president during the annual meeting in Tucson, professes to be a recycled auxilian. Starting her Auxiliary career in the early 60's, she served as state treasurer soon after Dr. Parsons opened his Tucson practice of Internal Medicine with a sub-specialty in Rheumatology. In the late 60's she worked actively in the Pima County Auxiliary and again related to the state board as county president. Having since been first vice president and president-elect, Carolyn comes to the state presidency qualified with Auxiliary background sandwiching a brief recess.

Her return to Auxiliary leadership status was largely motivated by her concern over the changing standards imposed upon medicine today and the image-modifying results to physicians. She feels committed as a doctor's wife to be supportive of her husband and is among those who are comfortable doing so without fear of losing personal identity.

A traditionalist at heart, Carolyn Parsons believes in the natural change which comes through Communication and Education - her priorities aimed at spreading awareness of the Auxiliary health programs. The same priorities are intended toward furthering legislative effort, understanding among members, and needs of the organization.

The non-abrasively direct, clear eyed president avows greater responsibility for accomplishment placed on persons chosen to Auxiliary positions in the belief that talents are unlimited and should be activated by incentive to carry on with creativity.

So thereby hangs the Arizona Auxiliary profile of the year ahead: positive, optimistic, and idealistic - surely an enviable pattern for all.

Mrs. Melvin W. (Jean) Phillips
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In Memoriam

James R. Atkinson

(January 6, 1929 - February 12, 1978)



Jim Atkinson and Lona, his wife for twenty years, lost their lives in their twin-engine Piper Comanche plane near the summit of Santiago Peak in the Santa Ana Mountains in California on Sunday evening, February 12, 1978. Both were expert flyers, having flown this plane to medical meetings in the United States, the Caribbean, Bermuda, England, Brazil, and, in 1975, around the world. At the time of his fatal accident Jim was on his way to meet with California engineers for the purpose of development of an intracranial pressure monitoring device, a project that he had been involved with for over ten years and was being funded by the Women's Board of the Barrow Neurological Foundation.

Doctor Atkinson was born in McGill, Nevada, received the B.S. degree in chemistry from the University of Nevada, and the M.D. degree from the University of Washington in Seattle in 1957. He developed a firm base in basic sciences prior to graduation from medical school, including Graduate School appointments at the University of Washington in psychology (1950-1952) and physiology (1955-1956). He expanded these interests with appointments as a trainee in biochemical genetics (1951-1952), neurophysiology (1952-1953), radioisotopes (1956), and biophysics (1953-1957). He was a neurosurgical resident at the University of Washington (1958-1962) under the direction of Arthur A. Ward, Jr., M.D., during which time he collaborated with Doctor Ward on the first published work on intracellular ultramicroelectrode techniques in epileptic foci, and, with Doctor Eldon Foltz, on the use of intraventricular RISA as a diagnostic aid in pre and post-operative hydrocephalus. During his time in Seattle Jim became an expert SCUBA diver and was the first to demonstrate air embolism in the brain under simulated conditions in the laboratory. His interests in pain and addiction were begun in the Pain Clinic at the University of Washington and with operations, including cingulumotomy, by Doctor Foltz. During 1962-1963 he worked with Doctor Ward in the area of evoked cortical unit recording and in cerebrospnal fluid dynamics, and was certified by the Atomic Energy Commission following work in radioisotope techniques at the U.S. Naval Medical School in Bethesda. He held N.I.H. post-doctoral fellowships from 1960-1963.

As the Barrow Neurological Institute of St. Joseph's Hospital and Medical Center was being developed as an academic and regional center for the neurological and neurosurgical specialities, Jim was selected to join the hospital-based group to add his skills, particularly from the standpoints of his research and educational capabilities. His subsequent performance during the past fifteen years proved the value of his preparation. He was certified by the American Board of Neurological Surgery in 1965. The Neurosurgical Research Laboratory of the Institute was founded in 1966 by Jim with a grant from the Women's Board of the Barrow Neurological Foundation. He also founded an interdisciplinary Pain Clinic the same year. Stereotactic clinical and research facilities were expanded by additional funding. He was generous with his time and talent in teaching both attending and resident staff members and provided these individuals with his help and that of the engineering staff upon request.

Jim was precise, completely honest and straight-forward, and earned the deep respect of his colleagues and residents. He gradually developed recognition for his work in the fields of cerebral blood flow, intracranial pressure monitoring, and pain. He, with Shurtleff and Foltz, published a fundamental work in the area of radiotelemetry for the measurement of intracranial pressure (J. Neurosurgery, 27:428-432, 1968), following his participation as

Visiting Scientist at the University of Washington for three months in 1966. He participated in many scientific meetings, locally, nationally, and abroad. Among the many neurosurgical residents whose lives were enriched by spending time at the B.N.I. with Jim, two should be mentioned. L. Philip Carter was the winner of the Western Neurosurgical Society Resident Award in 1972 because of work on cortical blood flow in controlled hypotension as measured by a thermal diffusion probe. The Atkinson-Carter Cerebral Blood Flow Probe has been further developed by Doctor Carter who has developed laboratory facilities in the Barrow Neurological Institute to supplement his clinical work. Andrew G. Shetter won the American Academy of Neurological Surgery Resident Award in 1975 based on work in the area of pain (Dorsal Column Stimulation's Effect of Bulboreticular Unit Activity Evoked by Noxious Stimuli). Doctor Shetter has worked with Doctor William H. Sweet of the Massachusetts General Hospital since July 1, 1976, in the clinic and laboratory, in the area of pain control. He has been selected to return to the B.N.I. as Director of the Neurosurgical Research Laboratory and to join the Division of Neurosurgery on a geographical full time basis. Obviously Doctor Atkinson has left a legacy of excellence worthy of emulation.

Jim Atkinson was a member of the Maricopa County and Arizona Medical Societies, the American Association of Neurological Surgeons, the American Academy of Neurological Surgery, the American Association for the Advancement of Medical Instrumentation, the Research Society of Neurological Surgeons, and the Western, Arizona, and Phoenix Neurosurgical Societies. He was the author of co-author of 26 scientific publications.

Jim was a most compassionate physician who looked after both the major and minor needs of his patients and their families. We have received hundreds of letters from his patients and friends, many from sources that none of us were aware of until after his death. He was dedicated not only to neurological surgery as a science but to the social service that he could achieve.

He was devoted to his personal and professional families. He is survived by his mother, Mrs. Russell Atkinson, of Port Angeles, Washington.

A James R. Atkinson Memorial Fund has been created as a remembrance of Jim's quiet, productive career and life in our midst. This Fund will be used to carry on and to advance this remarkable man's efforts to alleviate pain and suffering in the Pain Clinic and Laboratories that he founded.

John R. Green, M.D.

John C. Lincoln Hospital

Judy Roberts
Public Relations

John W. Kennedy, M.D., Editor

In the early 1920's, Miss Elizabeth Beatty, a retired stenographer, arrived in Arizona from Pennsylvania. One of the things that touched her most was the deplorable conditions under which many tuberculosis patients and others suffering various respiratory ailments were living. About 200 families were concentrated in a small community called Sunnyslope, a few miles north of Phoenix. Their homes were tents and dirt-floor shacks. Almost penniless, they had come from other states putting their faith in the curative powers of the dry desert air and plentiful sunshine.

Dr. Fred Holmes and Mrs. Marguerite Colley were the health agents who brought medicines to the indigent wayfarers and helped Miss Beatty to form the Desert Mission. By the late twenties, a clinic, nursery, recreation hall, library and church were constructed with donations from many interested parties.

A fire in 1942 destroyed several buildings and new construction began on a 20-acre plot of land purchased with a donation by Mr. and Mrs. John C. Lincoln. The site was between Dunlap and Hatcher Roads and Second and Third Streets, where the present hospital stands.

In 1947, the Roy Brooks Clinic was built and services increased with the help of Dr. Bertram L. Snyder of Phoenix and several nurses. Mr. H.F. Hancox came in '48 as the first Administrator and worked to establish the Desert Mission Convalescent Hospital, a 16-bed unit located east of the clinic. The two facilities were joined in 1954 and incorporated as the John C. Lincoln Hospital in honor of its chief sponsor and donor.

Expansion followed rapidly with the construction of a $200,000 surgical wing and physical therapy facilities. A Pediatric unit was added in 1959, bringing the total capacity to 50 beds. When the operating suite was completed, the hospital had 65 employees and 50 doctors; now there are over 500 employees and almost 600 physicians on the medical staff.

The present ultra-modern structure was dedicated in 1965 with a total of 131 beds combining the original wings with the four-story facility. New services included an obstetrical department and intensive care unit.

Work began on an emergency and outpatient department and in 1970 the Fourth Floor was opened adding 70 patient beds and one of the most sophisticated Coronary Care Units in the State. The total bed complement is now 202.

Today, John C. Lincoln Hospital is still growing. The Hospital recently completed Phase III of a $7 million construction project which had added two floors to the existing four-story structure and replaced the original hospital wings with a ground level and first floor expansion.

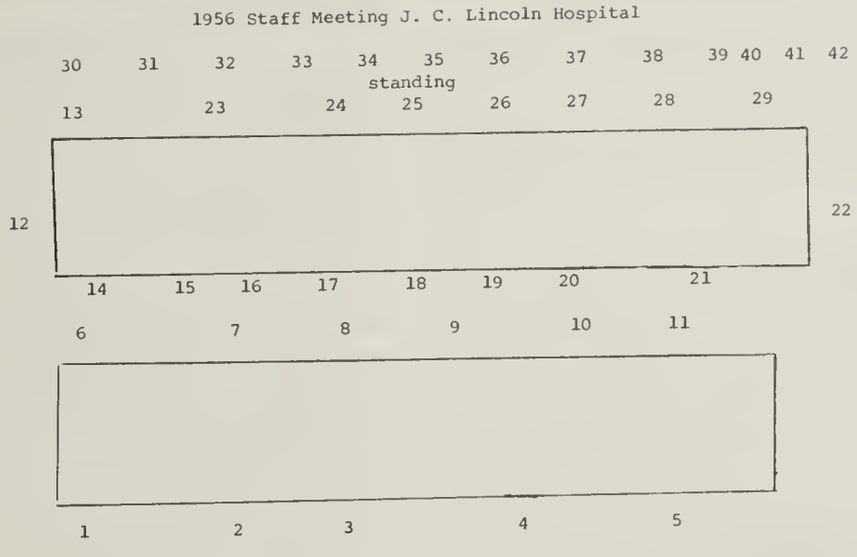
In January, 1976, the new Fifth Floor was opened with 54 general care patient beds and a 12-bed pediatric unit. The sixth floor is "shelled-in" with an eventual addition of 80 beds as need arises.

A new outpatient department including Outpatient Surgery, Cardiology, Respiratory Therapy and Occupational Therapy, opened in July of 1977. Relocation of the ancillary areas has provided more space for future growth and easy accessibility to the patient. The Radiology Department moved into the new wing, giving them an area three times larger (1978).

R.W. Leitner has served as Administrator since 1966 and Donald Davis, M.D., is currently Chief of the Medical Staff.

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.





1956 STAFF MEETING, J.C. LINCOLN HOSPITAL

1) Jacob Reichert; 2) Clyde Flood; 3) Francis Allinson; 4) George Page; 5) Ken Herbst; 6) Vic Mulligan; 7) Oscar Blende; 8) A.L. Rhodes; 9) Jack Herman; 10) Charles Lofdahl; 11) Sam Cohen; 12) Paul Jarrett; 13) Joe Likos; 14) Seymour Wiseman; 15) Gerold Hausbro; 16) Al Wagner; 17) Ted Price; 18) Herb Munhall; 19) Dale Stannard; 20) Dean Kovar; 21) Bob Beers; 22) H.F. Hancox (Admin.); 23) William Shike; 24) Manny Barnet; 25) Ord Shumway; 26) unknown; 27) Christian Risser; 28) Bill Rigley (Admin.); 29) Bert Snyder; 30) Stanley Holton; 31) Gordon Geisler; 32) Howard Kimball; 33) Robert Snapp; 34) Jack Vivian; 35) E.A. Henderson; 36) Fred Hodell; 37) Tracy Harold; 38) unknown; 39) Loy Roberts; 40) Don Fusco; 41) Charles Zusche; 42) Don Murray.

MEDICAL ECONOMICS COMMITTEE

Meeting of the Medical Economics Committee of the Arizona Medical Association, Inc., held Thursday, March 30, 1978 at 810 W. Bethany Home Road, Phoenix, Arizona convened at 7:45 p.m. Richard W. Switzer, M.D., Chairman, presiding.

PRESENTATIONS BY ICA

Mr. Robert Pointer, Management Analyst, Industrial Commission of Arizona, and Mr. Gregory Johnson, Assistant to the Director of ICA, presented methodology that the ICA utilized during the previous year in determining the fee schedules. Tables used in the explanation are on file at the Association headquarters.

ICA NEGOTIATIONS 1978

A draft of a proposal to present to the ICA during meeting scheduled for 10:00 a.m., Thursday, April 13, 1978 was discussed. There were three major sections to the proposal:

1. Revising the present conversion factors based on the currently used 1964 CRVS, 1973 ASA Guide, and 1973 ACR.
2. Converting from the 1964 CRVS to the 1974 CRVS and proposing appropriate conversion factor.
3. Reassessing the expert witness fees.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE INDUSTRIAL COMMISSION OF ARIZONA THAT WE CONTINUE UTILIZING THE 1964 CRVS and 1973 ACR AND TO CHANGE TO THE 1975 ASA GUIDE.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE INDUSTRIAL COMMISSION OF ARIZONA THAT EXPERT WITNESS FEES BE INCREASED TO $60.00 PER HOUR, MINIMUM OF 3 HOURS PER HEARING. FURTHER, THAT WHEN A HEARING IS CANCELED WITHIN 24 HOURS OF A HEARING THE MINIMUM WITNESS FEE OF $180.00 BE ALLOWED; WHEN A HEARING IS CANCELED WITHIN 48 HOURS OF THE SCHEDLED HEARING, ONE HALF THE MINIMUM WITNESS FEE BE ALLOWED.

IT WAS MOVED AND CARRIED THAT WE RECOMMEND A MINIMUM OF 7.7% INCREASE IN THE PRESENT CONVERSION FACTORS, ROUNDED TO THE NEAREST $.25. THE 7.7% INCREASE RESULTS FROM: A PROJECTED 6% INCREASE IN THE CONSUMER PRICE INDEX (CPI) FOR THE NEXT YEAR AND A 1.7% UNDERESTIMATION OF THE CPI FOR 1977 BASED ON OUR PROJECTED 4.97% INCREASE AND THE ACTUAL INCREASE OF 6.7%;

THUS THE PROPOSED MINIMUM CONVERSION FACTORS RECOMMENDED WOULD BE AS FOLLOWS:

Category	Present Conversion Factor 9/1/77	Proposed Conversion Factor 9/1/78
Medicine	$11.50	$12.00
Surgery	9.75	10.00
Laboratory	7.25	7.75
Radiology		
Total Service	8.25	9.00
Professional Component	2.00	2.25
Anesthesiology	12.50	13.50

IT WAS MOVED AND CARRIED THAT WHILE IT IS RECOGNIZED THAT THE ABOVE PROPOSED CONVERSION FACTORS ARE LESS THAN THE CURRENT FEES CHARGED BY THE MAJORITY OF PHYSICIANS IN ARIZONA, MAXIMUM EFFORT BE GIVEN TO BRING THE FEE SCHEDULES BY THE INDUSTRIAL COMMISSION OF ARIZONA UP-TO-DATE WITH CURRENT CHARGES. TO ACCOMPLISH THIS END, COOPERATION SHOULD BE SOUGHT FROM THIRD PARTY CARRIERS, I.E. BLUE SHIELD AND MEDICARE TO INDICATE THE CURRENT PREVAILING PHYSICIAN CHARGES.

Quality Control - Discussion ensued concerning the present system of collection and retrieval of data by I.C.A. It would appear that they are recipients of a great deal of information if it were properly organized. It was determined that the Association would stand ready to provide assistance in developing parameters for the collection of data and interpretation of the results and that this matter be made part of our presentation to I.C.A.

It was determined that Drs. Switzer, Dexter, Moraca and Bennett would form the official team to visit with the Industrial Commission on April 13th with Mr. Beckman and Mr. Barnett.

LEGISLATIVE COMMITTEE

The meeting of the Legislative Committee of the Arizona Medical Association, Inc., held Saturday, March 4, 1978 at 810 West Bethany Home Road, Phoenix, Arizona, convened at 1:28 p.m., Selma E. Targovnik, M.D., Chairperson, presiding.

LEGISLATIVE REVIEW

S.B. 1110 Smoking - This bill would provide that smoking would be prohibited in certain public places, i.e., grocery, drug or department stores.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT S.B. 1110 PROHIBITING THE SMOKING OF TOBACCO IN CERTAIN PLACES.

H.B. 2193 - Abortion; Informed Consent - H.B. 2193 would provide physicians must give an explanation prior to the abortion reasonably sufficient to apprise the patient of all material medical information concerning the procedure and its probable psychological effects, outlining the contents of the explanation and providing a maintenance of records.

IT WAS MOVED AND CARRIED TO NON-SUPPORT H.B. 2193 PROVIDING FOR AN INFORMED CONSENT FOR ABORTIONS.

H.B. 2165 Abortion; Information Report - This legislation provides that any person who performs an abortion shall provide in a report considerable information to the Department of Health Services within a period of six (6) months.

IT WAS MOVED AND CARRIED TO OFFER NON-SUPPORT TO H.B. 2165.

H.B. 2145 Abortion; Notification to Parents.

IT WAS MOVED AND CARRIED TO NON-SUPPORT H.B. 2145 WHICH PROVIDES FOR PARENTAL AND SPOUSAL NOTIFICATION OF ABORTIONS.

H.B. 2132 Abortion; Use of Public Funds - This legislation prohibits the use of public funds for abortion of any political subdivision of the State.

IT WAS MOVED AND CARRIED TO OFFER GENERAL NON-SUPPORT TO H.B. 2132.

H.B. 2115 Abortion; Certificate of Fetal Death - H.B. 2115 would provide for the filing of a certificate of fetal death by abortion; it would prescribe the contents in the certificate and a system of monthly reporting by local registrars and the Department of Health Services.

IT WAS MOVED AND CARRIED TO NON-SUPPORT H.B. 2115 PROVIDING FOR A CERTIFICATE OF FETAL DEATH.

H.C.R. 2013 Constitutional Amendment - Right to Life - It is proposed that an amendment to be made to the constitution of the United States to protect the right to life of all human beings.

IT WAS MOVED AND CARRIED TO OFFER NON-SUPPORT TO H.C.R. 2013 PROVIDING FOR A CONSTITUTIONAL AMENDMENT; RIGHT TO LIFE.

S.B. 1106 Birth and Death Registrations - This legislation was introduced at the request of the Department of Health Services prescribing registration for births in a moving conveyance and that a fetal death certificate would be required after a gestation period of twenty (20) completed weeks or more, *or* if the fetal remains weigh 350 grams or more.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT S.B. 1106 PROVIDING FOR AN ADDITIONAL CIRCUMSTANCE FOR FETAL DEATH REGISTRATION.

S.B. 1379 Study Council - S.B. 1379 would provide for a study council on health care laws and regulations preparing a comprehensive final report on findings and recommendations to the Governor. President of the Senate, Speaker of the House on or before January 1, 1979 with an appropriation of $1,000.00

IT WAS MOVED AND CARRIED TO TAKE NO ACTION ON S.B. 1379 PROVIDING FOR A STUDY COUNCIL ON HEALTH CARE.

H.B. 2268 Minors Consent; Drug Use - H.B. 2268 would provide for the capacity of a minor to consent to treatment of alcohol use. This is in addition to statutes provided for minors consent to treatment for use of a dangerous drug or narcotic. This legislation further provides that the parent of the child shall be notified and that unless expressly consented to, no assessments for treatment shall be levied against the parents.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT THE CONCEPT OF H.B. 2268 PROVIDING THAT THE PROVISIONS IN THE LEGISLATION PROVIDING FOR NOTIFICATION TO THE PARENT AS WELL AS THE ASSESSMENT FOR TREATMENT PORTIONS ARE REMOVED.

H.B. 2299 Immunity - Blood Alcohol Tests - H.B. 2299 provides for an immunity to physicians, nurses and qualified technicians for drawing blood at the request of law enforcement officers for blood alcohol testing purposes. It was noted that the current legislation provides for immunity of acts without malice and the physician/patient privilege would be non applicable when chemical test results are in issue or relevant.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT THE CONCEPT OF H.B. 2299 WITH THE PROVISO THAT THE PHYSICIAN/PATIENT PRIVILEGE BE CLARIFIED AND THAT THE STATE, COUNTY OR OTHER DIVISION DEFEND THE PHYSICIAN.

H.B. 2290 Requests for and Collection of Specimens and Human Blood; Clinical Labs - This bill would provide that the person being tested could authorize the collection of specimen or human blood as well as authorize the clinical lab to examine such specimen.

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT H.B. 2290.

S.B. 1104 Clinical Laboratory Licensure Exemption - The Department of Health Services requested legislation to exempt certain classes of clinical labs from licensure such as those performing tests and procedures solely for eligibility of insurance coverage and public health programs or projects under contract or grant from a governmental entity. Also allows paramedics who have written approval of a director to collect blood under the direction or request of a licensed physician.

IT WAS MOVED AND CARRIED TO GENERALLY SUPPORT S.B. 1104 PERTAINING TO CLINICAL LABS EXEMPTIONS.

H.B. 2231 Ownership of Health Records - This legislation would provide that the patient owns the medical records.

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT H.B. 2231 PERTAINING TO OWNERSHIP OF HEALTH RECORDS.

S.B. 1165 State Hospital Superintendent - This legislation would provide that the director of the Department of Health Services shall accede to certain duties of the superintendent of the State Hospital. It was reported that the Arizona Psychiatric Society was in the process of working with the Director of the Department of Health Services concerning the duties of the superintendent of the State Hospital and that it may not be appropriate to adopt this legislation at this time.

IT WAS MOVED AND CARRIED TO OFFER NON-SUPPORT TO S.B. 1165 CONCERNING THE SUPERINTENDENT OF THE STATE HOSPITAL.

S.B. 1356 Statewide Health Coordinating Council (93-641) - This legislation would conform Arizona with the Federal law 93-641 providing for a statewide health coordinating council prescribing powers, duties and planning functions.

IT WAS MOVED AND CARRIED TO OFFER ACTIVE NON-SUPPORT TO S.B. 1356 CONFORMING ARIZONA WITH THE FEDERAL LAW 93-641.

S.B. 1306 Licensing of Accredited Hospitals - The Arizona Hospital Association introduced legislation which would provide for the ability of the Department of Health Services to accept the accreditation of the joint commission

accreditation of hospitals or the American Osteopathic Association thereby saving the time and effort of dual surveys or inspections.

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT THE ENACTMENT OF S.B. 1306.

H.B. 2303 Hospital Facilities Commission - Representatives McCarthy and Flynn introduced legislation which would provide a hospital facility commission prescribing membership, organization, powers and duties. The primary purpose of the commission would be to review the rates of hospitals and perhaps determine certificate of need. These duties currently rest with the Health Systems Agency and are regulatory powers rather than planning.

IT WAS MOVED AND CARRIED TO OFFER GENERAL SUPPORT OF THE CONCEPT OF A HOSPITAL FACILITIES COMMISSION.

H.B. 2337 Emergency Medical Services -Currently the Emergency Medical Services reside in four state agencies; D.H.S., D.P.S., D.O.T., and the Corporation Commission. The original intent of H.B. 2337 was to bring the Emergency Medical Services under one department and to provide for medical control. It was reported that it does not appear to be politically feasible to accomplish that goal, therefore it is intended to provide for a medical director with appropriate medical control outlined in the statutes as well as a medical advisory committee.

IT WAS MOVED AND CARRIED TO OFFER GENERAL SUPPORT TO H.B. 2337.

H.B. 2403 & S.B. 1347 Indigent Health Care - Legislation has been introduced in both Houses which would provide for a standardization of health care for the indigent throughout the fourteen (14) counties. The benefit structure is patterned primarily after Maricopa County. It also provides for a new definition of indigent for health services as well as a provision for state payment of indigent of health. It was reported that this legislation was drafted by the Council on County Governments.

IT WAS MOVED AND CARRIED TO OFFER GENERAL SUPPORT TO H.B. 2403 AND S.B. 1347 PROVIDING FOR INDIGENT HEALTH CARE.

H.B. 2169 Catastrophic Health Benefits Protection - This legislation will provide for comprehensive catastrophic health benefit protection through a fund to be administered by the insurance director. It establishes an assigned risk plan to make available insurance to the uninsurables as well as the near uninsurables.

IT WAS MOVED AND CARRIED TO OFFER GENERAL SUPPORT TO THE CONCEPT OF A STATE CATASTROPHIC HEALTH INSURANCE BENEFIT PROGRAM.

S.B. 1252 Medical Student Loans - This legislation would provide for an appropriation of $60,000 to the Board of Medical Student Loans adopted during the last legislative session.

IT WAS MOVED AND CARRIED TO OFFER ACTIVE SUPPORT OF MEDICAL STUDENT LOAN APPROPRIATION.

S.B. 1132 Removing C.M.E. Requirements - This legislation would remove requirements for continuing education as a condition for renewal of licensure from all professional and occupational acts including the Medical Practice Act. Mr. Barnett reported that the legislation had already been heard and failed to pass the Health and Welfare Committee by a margin of 9-1. NO ACTION TAKEN.

S.B. 1323 Council for the Deaf - During the last session, legislation was enacted providing for a council for the deaf. Within the legislation was the requirement that a physician report the name and address of a deaf person to the Council. This provision is in direct opposition to the doctor/patient privilege and legislation was introduced to strike this mandatory reporting feature.

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT S.B. 1323 REMOVING MANDATORY REPORTING PROCEDURE FROM THE REGISTER.

S.B. 1353 Communication Disorders Services - This legislation would provide for licensure of Audiologists and speech pathologists and creates a ninety-ten licensing board.

Dr. Richard D. Zonis, President of the State Society of Otolaryngology presented a letter concerning this legislation. He noted that "I don't know of any segment of the public which is being harmed by any such abuses, and I don't particularly see the need for a licensing bill".

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT S.B. 1353 CREATING A BOARD OF COMMUNICATION DISORDERS.

S.B. 1351 Homeopathic Physicians - This legislation would exempt from the Medical Practice Act homeopathic physicians.

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT S.B. 1351 EXEMPTING FROM THE MEDICAL PRACTICE ACT HOMEOPATHIC PHYSICIANS.

S.B. 1344 Naturopathy Act - S.B. 1344 was introduced at the request of the Naturopathic Association and would generally update their act. Concerns were expressed that any updating of the naturopathic act simply gives more credence to them and especially when they ask for educational qualifications to do emergency procedures and minor surgery.

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT S.B. 1344 UPDATING THE NATUROPATHY ACT.

S.B. 1350 Optometry Act - S.B. 1350 provides for updating certain provisions in the Optometric Act.

IT WAS MOVED AND CARRIED TO TAKE NO ACTION ON S.B. 1350 PROVIDING FOR UPDATING THE OPTOMETRIC ACT.

H.B. 2283 - S.B. 1333 Updating Psychologist Act - Two different pieces of legislation updating the psychologist practice act were reviewed.

IT WAS MOVED AND CARRIED TO TAKE NO ACTION ON EITHER H.B. 2283 or S.B. 1333 UPDATING THE PSYCHOLOGIST PRACTICE ACT.

H.B. 2339 Osteopathic Practice Act - Dr. McGill reported that the Osteopathic Medical Association had requested introduction of legislation to put the Osteopathic Practice Act in conformance with the Medical Practice Act.

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT LEGISLATION UPDATING THE OSTEOPATHIC MEDICAL PRACTICE ACT.

S.B. 1349 Physician's Assistants - Mr. Boykin informed the Committee that they are currently having some difficulties with the Physician's Assistants Act as enacted during the last legislative session. The first problem being that there was not an appropriation. Second that even if there was an appropriation and monies were collected, there seems to be some question as to whether the Joint Board of Medical and Osteopathy Examiners has the authority to expend the funds. The final problem is whether the Board of Medical Examiners' personnel can provide services necessary to carry out the purposes of the Physician's Assistants law. All of these developments have just arisen and therefore it will be necessary to confer with legal council to correct any problems through legislation.

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT LEGISLATION WHICH WILL ALLOW THE ADMINISTRATION OF THE PHYSICIAN'S ASSISTANTS ACT AS ORIGINALLY INTENDED.

S.B. 1427 Chiropractic and Naturopathic Insurance Benefits - This legislation was introduced with the permission of the Committee on Rules three weeks after the due date for introduction, and provides that any covered service performed by a Chiropractor or Naturopath within the lawful scope of such person's license would not be denied by insurance companies.

IT WAS MOVED AND CARRIED TO ACTIVELY NON-SUPPORT S.B. 1427 PROVIDING THAT BENEFITS SHALL NOT BE DENIED FOR COVERED SERVICES PERFORMED BY CHIROPRACTIC AND NATUROPATHIC PHYSICIANS.

H.B. 2338 Professional Liability Insurance - This legislation would provide that professional liability would be covered by the Arizona Property and Casualty Guarantee Fund, and amending the definition of a licensed health care provider at the request of the J.U.P.

IT WAS MOVED AND CARRIED TO ACTIVELY SUPPORT H.B. 2338.

H.B. 2415 P.K.U. Testing - Representative Kunasek asked that the Association review H.B. 2415 which provides for the attending physician to state on the birth certificate the results of tests for metabolic disorders which shall include a test for P.K.U. and Hypothyroidism. The committee recalled the action taken during the last meeting which was in cooperation with the Department of Health Services, the State Pediatric Society, legislation would be drafted for introduction during the next session which would satisfy the intent of H.B. 2415.

IT WAS MOVED AND CARRIED TO REAFFIRM THE COMMITTEE'S ACTION DURING ITS LAST MEETING CONCERNING METABOLIC SCREENING.

Meeting Adjourned 3:56 p.m.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona on Saturday, February 25, 1978, a quorum being present, convened at 9:08 a.m., John F. Kahle, M.D., President and Chairman, presiding.

PERCENTAGE LEASES

Mr. Jacobson reviewed an extensive opinion concerning "the legality of percentage leases for hospital based physicians" dated February 10, 1978. He stated, in part, that "based upon our examination, it is the opinion of this office that, in the future, percentage arrangements of any kind between physicians and hospitals, at the very least, are not free from substantial risk and, at the most, might well constitute acts of unprofessional conduct under Arizona law. The entire text of the opinion is on file in the Association headquarters office.

IT WAS MOVED AND CARRIED TO ACCEPT THE LEGAL OPINION BY THE FIRM OF SNELL & WILMER CONCERNING "THE LEGALITY OF PERCENTAGE LEASES FOR HOSPITAL BASED PHYSICIANS"; TO CIRCULATE IT IN AN EXERPT FORM THROUGH MEDICAL MEMOS; TO SEND IN ITS ENTIRETY TO ALL MEMBER PATHOLOGIST AND RADIOLOGIST AND OTHERS WHO REQUEST IT.

JEPPESEN SANDERSON - MED-PREP

Mr. Donald R. Sellars, Manager, Training Systems Design of Jeppesen Sanderson, a Denver based subsidiary, The Times Mirror Company, presented the Med-Prep program developed by them to the Board. Med-Prep is an acronym for Medical Patient Risk Education Program designed to assist physicians to affectively prepare patients for medical and surgical procedures with audio-visual presentations currently covering sixteen specialties. The films explain the procedure and its potential risk with possible alternative courses of treatment in lay language. The program is being developed in cooperation with the Colorado Medical Society. It was proposed that the Association become a distributor of the Med-Prep program.

IT WAS MOVED AND CARRIED TO THANK JEPPESEN SANDERSON FOR THEIR PRESENTATION TO THE ASSOCIATION.

MULTI-STATE PHYSICIAN PURCHASING SERVICE

Mr. Robert G. Smith, Executive Director of the Wyoming State Medical Association, presented a proposal to the Board to develop a multi-state physician purchasing service organization. He stated that from our investigation and follow-up survey, it would appear that a joint purchasing program for physicians through the Western State Medical Associations would be feasible. The states interested in such a program are Colorado, Montana, Idaho, Oregon, New Mexico, Arizona and Wyoming.

"**Summary**

One thing is essential to the operation of such a program — WE WOULD HAVE TO BE ABLE TO DELIVER THE ESTIMATED SALES TOTAL. If we did not do this consistently, few suppliers would sign agreements with us.

"It is somewhat difficult to ascertain a typical amount of purchases for a physician. It varies widely and depends on the specialty, the type of practice, rural or urban location, proximity to a hospital, etc. However, our spot-checks show a physician will probably spend between $4,000 and $6,000 on drugs and supplies — not counting major medical equipment and office equipment purchases. This total, of course, also depends of the breadth of your definition of supplies.

"It is also important to note that in states where the hospitals do not have purchasing groups, there seems to be some interest in joining a physicians' purchasing program. This addition would boost projected sales totals substantially.

"The Wyoming State Medical Society would be interested in having the administrative offices for a possible program in Wyoming and are willing to allow their Society's executive director some time in working to create such a program.

"We recommend that if a purchasing program is formed, it only be handled through the state medical associations — and only those associations which endorse the program.

"The ingredients for a physicians' joint purchasing program are at hand in the western states. Such a program would provide additional tangible benefits to members of a state medical association. Such a program could save physicians in private practice in our states millions of dollars over a period of time. On an individual basis, there seems to be no major barrier to saving a minimum of 18 per cent in overall purchases of supplies, drugs and equipment.

"We must decide now whether a joint purchasing program is accomplishable, of great enough interest to our members and in the best interests of our associations.

"**PROJECTIONS**

Service begins: January 1, 1979

Initial billing period: January - December, 1979

NOTE: Billing to be on voluntary basis as part of State Medical Association dues statements.

Start-up operation begin: July 1, 1978

Total Potential Market: 12,600 members (7 States)

ANNUAL PURCHASING SERVICE CHARGES

Solo Practitioner	$50.00
Groups	
First physician	40.00
Second physician	35.00
All other members	25.00 each

PROJECTED INCOME (1979)

3,200 members at $50	$160,000
1,000 members (First member - group) at $40	40,000
1,000 members (Second member - group) at $35	35,000
1,000 members (Other group members) at $25	27,500
6,300 Members (50% of Total Members)	$262,500

PROJECTED EXPENDITURES (1979)

$ 89,000	- Repayment (principal and interest) to State Medical Associations.
$ 52,500	- Rebate to State Medical Associations ($10 each for $50 memberships, $8 each for $40 memberships, $7 each for $35 memberships, $5 each for other memberships)
$121,000	- Operating funds - 1979
$262,500	- TOTAL

"**FINANCING START-UP OF PURCHASING SERVICE**

"Approximately six months is needed to organize purchasing service, negotiate contracts and market purchasing service to physicians in the various states.

"Maximum costs for the six-month start-up would total $83,200.

"To raise these funds, we propose that each participating State Medical Association loan the new purchasing service $12,000 at 7% annual interest for a period of nine to twelve months.

"Assuming accomplishment of the purchasing service's minimum goals, these loans plus interest would be paid back to the State Medical Associations by March, 1979."

IT WAS MOVED AND CARRIED TO ADVANCE THE $12,000 TO ORGANIZE THE PURCHASING SERVICE PROGRAM AT 7% ANNUAL INTEREST FOR A PERIOD OF 9-12 MONTHS IN ORDER TO GET THE PROGRAM STARTED.

AUXILIARY REPORT

A letter was received from Mrs. James L. Parsons, President Elect, Arizona Medical Association Auxiliary, Inc. in appreciation for the grant of $2,400. Mrs. Parsons informed the Board that during her year as President, she plans to emphasize immunization, VD, quackery, CPR and breast self examination. The Arizona Medical Association Auxiliary is the link of education in these areas to the counties and it is the counties who will inturn project programs as needed in their own areas. RECEIVED FOR INFORMATION.

A.H. ROBINS 1978 PHYSICIAN AWARD FOR COMMUNITY SERVICES

Written presentations were made by the following county societies nominating physicians for the A.H. Robins 1978 community services award:

1. Coconino County Medical Society nominates J. Charles Casebeer, M.D.
2. Maricopa County Medical Society nominates H. Belton P. Meyer, M.D.
3. Pima County Medical Society nominates Hugh C. Thompson, M.D.
4. Yuma County Medical Society nominates Paul J. Slosser, M.D.

BY SECRET BALLOT IT WAS DETERMINED THAT H. BELTON P. MEYER, M.D., WOULD BE THE RECIPIENT OF THE 1978 A.H. ROBINS PHYSICIAN AWARD FOR COMMUNITY SERVICES.

AMA NOMINATIONS FOR AWARDS

Joseph A. Goldberger Award

IT WAS MOVED AND CARRIED TO NOMINATE GLEN A. FREIDMAN, M.D. FOR THE JOSEPH A. GOLDBERGER AWARD IN CLINICAL NUTRICIAN FOR HIS DEVELOPMENT OF THE HEALTH EVALUATION AND LONGEVITY PLANNING FOUNDATION (HELF).

The Arnold and Marie Swartz Award

IT WAS MOVED AND CARRIED TO NOMINATE TO THE AMERICAN MEDICAL ASSOCIATION H. BELTON P. MEYER, M.D. FOR THE ARNOLD AND MARIE SWARTZ AWARD IN MEDICINE FOR HIS OUTSTANDING WORK IN THE CARE AND SURVIVAL OF HIGH RISK INFANTS IN ARIZONA AS WELL AS HIS EFFORTS IN DEVELOPING THE ARIZONA PERINATAL PROGRAM.

BENEVOLENT & LOAN FUND COMMITTEE

Medical Student Loan Fund Advisory Board

As a result of legislation adopted during the last session, a medical student loan fund advisory board has been created. The Board of Medical Examiners appointed Arthur V. Dudley, Jr., M.D. and Bruce N. Curtis, M.D. as their representatives to the Board. RECEIVED FOR INFORMATION.

EXECTIVE COMMITTEE

Membership Classification Changes Approved

Coconino County Medical Society

Rebecca Hildebrand, M.D. - Active to Associate - Account Residency - Dues Exempt - effective 1/1/78.

Kirk R. Stetson, M.D. - Requests Dues Exemption for 1977 - Account Financial Hardship.

Wesley E. Green, M.D. - Requests Dues Exemption for 1977 - Account Financial Hardship.

Maricopa County Medical Society

B.J. Axel, M.D. - Active to Associate - Account Illness - Dues Exempt - Effective 1/1/78. Also requests dues exemption for 1977 - Account Illness.

Robert D. Barker, M.D. - Active to Associate - Account Retirement - Dues Exempt - Effective 1/1/78.

Matthew Cohen, M.D. - Active to Active Over 70 - Account Age - Dues Exempt - Effective 1/1/78.

E.S. Cronk, M.D. - Active to Associate - Account Illness - Dues Exempt - Effective 1/1/78.

Richard E.H. Duisberg, M.D. - Active to Over 70 - Account Age - Dues Exempt - Effective 1/1/78.

Gordon Geisler, M.D. - Active to Associate - Account Retirement - Dues Exempt - Effective 1/1/78.

Louis C. Kossuth, M.D. - Active to Associate - Account Retirement - Dues Exempt - Effective 1/1/78.

Sam M. Mackoff, M.D. - Active to Associate - Account Illness - Dues Exempt - Effective 1/1/78.

Olga Quitmeyer, M.D. - Requests Dues Exemption for 1977 and 1978 - Account Financial Hardship.

Ronald M. Tolls, M.D. - Active to Affiliate - Account Out of State - Dues Exempt - Effective 1/1/78.

Murray G. Urie, M.D. - Active to Associate - Account Retirement - Dues Exempt - Effective 1/1/78.

Stewart Van Hoosear, M.D. - Requests Dues Exemption for 1977 - Account Financial Hardship.

Pima County Medical Society

Hollis H. Brainard, M.D. - Active to Associate - Account Retired - Dues Exempt - Effective 1/1/78.

Harold F. Stolz, M.D. - Active Over 70 to Associate - Account Retired - Dues Exempt - Effective 1/1/78.

William C. Bucher, M.D. - Active to Affiliate - Account Moved Out of State - Dues Exempt - Effective 1/1/78.

Terry Wood, M.D. - Active to Affiliate - Account Moved Out of State - Dues Exempt - Effective 1/1/78.

Andrew W. Nichols, M.D. - Active to Affiliate - Account Out of State - Dues Exempt - Effective 1/1/78.

Bernard M. Kuhr, M.D. - Active to Affiliate - Account Out of State - Dues Exempt - Effective 1/1/78.

Sidney R. Kimberling, M.D. - Request for Dues Exemption for 1978 - Account Financial Hardship.

Santa Cruz County Medical Society

Juan S. Gonzalez, M.D. - 50 Year Club to Associate - Account Retirement - Dues Exempt Effective 1/1/78.

FINANCE COMMITTEE

Resolution - 1979 Calendar year Budget of Income and Expense

The following resolution was presented to the Board of Directors for consideration to introduce to the House of Delegates during annual meeting 1978:

WHEREAS, It is customary for the House of Delegates to approve the Budget of Income and Expenditures for the next succeeding calendar year of the Arizona Medical Association, Inc.; therefore be it

RESOLVED, That the following Budget of Income and Expenditures for the calendar year 1979 be adopted:

	INCOME	EXPENDITURES	
General Dept.	$595,605.00	$578,400.00	$ 17,205.00
Committee Dept.	83,100.00	108,100.00	(25,000.00)
Annual Meeting Dept.	60,600.00	56,640.00	3,960.00
Publishing Dept.	72,470.00	68,430.00	4,040.00
TOTAL	$811,775.00	$811,570.00	$ 205.00

; and be it further

RESOLVED, That the annual dues of the Arizona Medical Association, Inc. be $250 for Active members, with $230 going to General Department Budget and $20 going to the Publishing Department Budget; and be it further

RESOLVED, That the Service member dues be $62.50 with $42.50 going to the General Department Budget and $20 going to the Publishing Department Budget; and be it further

RESOLVED, That the Intern or Resident Active member dues be $25 with $5 going to the General Department Budget and $20 going to the Publishing Department Budget.

IT WAS MOVED AND CARRIED TO INTRODUCE THE 1978 CALENDAR BUDGET OF INCOME AND EXPENSE TO THE HOUSE OF DELEGATES AS RECOMMENDED BY THE FINANCE COMMITTEE.

FINANCIAL STATEMENT

The Certified Public Account firm of Henry & Horne informed the Board of Directors that "we have examined the balance sheet of The Arizona Medical Association, Inc. as of Decembe 31, 1977 and 1976, and the related statement of operations, changes in fund balances and changes in financial position for the years then ended. Our examination was made in accordance with generally accepted auditing standards and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances.

"In our opinion the aforementioned statements present fairly the financial position of the Association at December 31, 1977 and 1976 and the results of its operations and the changes in its financial position for the years then ended in conformity with generally accepted accounting principles applied on a consistent basis.

"Our examination was made primarily to form an opinion on the basic financial statements. We also examined the accomanying departmental statement of general operations, building operations and benevolent and loan operations for the years ended December 3, 1977 and 1976. In our opinion, these supplemental departmental statements are fairly stated in all material respects as they relate to the financial statements taken as a whole."

February 17, 1978

The balance sheet and comany materials are on file in the Association's office. RECEIVED FOR INFORMATION.

POLICY RE: PRESIDENT - PRESIDENT-ELECT BUDGET

During the Finance Committee's review of the proposed 1979 budget, concern was expressed about providing a larger budget for the President and a new budget for the President-Elect. The Finance Committee recommends to the Board of Directors that they consider changing the present policy to one that would provide the President's budget of up to $7,500.00 a year and provide the President-Elect a budget of up to $2,500.00 per year.

IT WAS MOVED AND CARRIED TO PROVIDE THE PRESIDENT A BUDGET OF UP TO $7,500.00 AND PROVIDE THE PRESIDENT-ELECT A BUDGET OF UP TO $2,500.00 PER YEAR AS REIMBURSEMENT OF EXPENSES.

HEALTH MANPOWER COMMITTEE

PHYSICIAN'S ASSISTANTS AND NURSE PRACTITIONERS UNDER BOMEX

During meeting held January 11, 1978 the Health Manpower Committee reviewed H.B. 2003 introduced in the Arizona House of Representatives which would, if passes, allow nurse practitioners to prescribe as well as perform minor surgery. The following recommendations of the Health Manpower Committee were considered by the Board of Directors.

IT WAS MOVED AND CARRIED TO RECOMMEND THAT THE ARIZONA MEDICAL ASSOCIATION ACTIVELY OPPOSE H.B. 2003 TO THE UTMOST EFFORT THAT CAN BE EXERTED. (IT WAS NOTED THAT THE LEGISLATIVE COMMITTEE ACTIVELY OPPOSE H.B. 2003).

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE ARIZONA MEDICAL ASSOCIATION PROPOSE LEGISLATION TO PLACE ALL PHYSICIAN'S ASSISTANTS AND NURSE PRACTITIONERS IN THE EXTENDED ROLE, REGARDLESS OF THE TITLE THEY MIGHT USE, UNDER THE JURISDICTION OF THE JOINT BOARD OF MEDICAL EXAMINERS AND OSTEOPATHIC EXAMINERS IN MEDICINE AND SURGERY.

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION PROPOSE LEGISLATION TO PLACE ALL PHYSICIAN'S ASSISTANTS AND NURSE PRACTITIONERS IN THE EXTENDED ROLE, REGARDLESS OF THE TITLE THEY MIGHT USE UNDER THE JURISDICTION OF THE JOINT BOARD OF MEDICAL EXAMINERS AND OSTEOPATHIC IN MEDICINE AND SURGERY.

REGULATIONS FOR ARIZONA PHYSICIAN'S ASSISTANTS PROGRAMS

The Health Manpower Committee reviewed the regulations for Physician's Assistants recently adopted by the Joint Board of Medical Examiners and Osteopathic Examiners in Medicine and Surgery, and recommend that the Board of Directors take a favorable position regarding regulations.

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION ENDORSE THE RULES AND REGULA-

TIONS ADOPTED FOR THE ARIZONA PHYSICIAN'S ASSISTANTS PROGRAM BY THE JOINT BOARD OF MEDICAL EXAMINERS AND OSTEOPATHIC EXAMINERS IN MEDICINE AND SURGERY.

SEVENTH ANNUAL ARIZONA RURAL HEALTH CONFERENCE

The Health Manpower Committee reviewed the proposed program for the Seventh Annual Arizona Rural Health Conference and budget for the program and recommend to the Board of Directors that the Arizona Medical Association contribute $1,000.00 to the conference scheduled for May 12 and 13, 1978.

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION, INC. CONTRIBUTE $1,000 TO THE SEVENTH ANNUAL ARIZONA RURAL HEALTH CONFERENCE SCHEDULED FOR MAY 12 AND 13, 1978.

LEGISLATIVE COMMITTEE

STATUS REPORT

Mr. Barnett reviewed the actions of the Association's Legislative Committee and current status of legislation considered important to the Association such as the Advertising Bill, Optometry, Repeal of Medicaid and Substitution for Generics legislation.

CONFIRMATION OF APPOINTMENT

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF MR. NEIL WHITE, CLASS OF 1981 UNIVERSITY OF ARIZONA, COLLEGE OF MEDICINE AS A STUDENT MEMBER TO THE LEGISLATIVE COMMITTEE.

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF ROY G. BUBECK, D.O. AS REQUESTED BY THE ARIZONA OSTEOPATHIC MEDICAL ASSOCIATION TO REPLACE RATIBOR PANTOVICH, D.O. ON THE LEGISLATIVE COMMITTEE.

MATERNAL & CHILD HEALTH CARE COMMITTEE

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF ALBERT M. SCHWARTZ, M.D. TO THE MATERNAL & CHILD HEALTH CARE COMMITTEE.

MEDICAL EDUCATION COMMITTEE

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF MONT SMITH, M.D. AS A MEMBER OF THE MEDICAL EDUCATION COMMITTEE.

NATIONAL COUNCIL OF STATE CHAIRMEN OF CONTINUING MEDICAL EDUCATION

The Medical Education Committee informs the Board that "this newly formed organization was originally suggested over a year ago and has been in informative stages during the past year. It is intended as a forum where the persons directly involved with C.M.E. in each State Medical Association can meet and act in their mtual interest." Proposed bylaws of this organization which outlined the purposes and proposed activities were made available to the Board. The Medical Education Committee recommends support of this organization and that the Arizona Medical Association participate as a member with approval of an expenditure of $200.00 annually to maintain the membership.

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION, INC. SUPPORT THE NATIONAL COUNCIL OF STATE CHAIRMEN OF CONTINUING MEDICAL EDUCATION AND TO APPROVE THE EXPENDITURE OF $200.00 ANNUALLY TO MAINTAIN THE MEMBERSHIP.

PHYSICIAN REHABILITATION COMMITTEE

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF VICTOR J. ZANNIS, M.D. TO THE PHYSICIAN REHABILITATION COMMITTEE.

PROFESSIONAL COMMITTEE

ADVANCED CARDIAC LIFE SUPPORT PROGRAM

Dr. Kahle informed the Committee that the AMES Board of Directors has approved funding of the equipment peviously requested of ArMA for the Advanced Cardiac Life Support Program. Funding of some $13,000 will allow the program to start the training of physicians in this worthwhile project. It was further noted that the ArMA facilities will be utilized during the month of March for this purpose. RECEIVED FOR INFORMATION.

PUBLIC RELATIONS COMMITTEE

IT WAS MOVED AND CARRIED TO CONFIRM THE APPOINTMENT OF MARSHALL B. BLOCK, M.D. TO THE PUBLIC RELATIONS COMMITTEE.

AD HOC COMMITTEE ON U.S. SUPREME COURT DECISION REGARDING ADVERTISING

The Ad Hoc Committee on the U.S. Supreme Court Decision Regarding Advertising recommends to the Board of Directors that the Association propose legislation during this session of the legislatue to amend the medical practice act by providing for a new definition of advertising under professional conduct to read: "advertising a false, deceptive or misleading manner, advertising the quality of medical services, advertising by in-person solicitation and advertising procedures and practices which are illegal." The Board was informed that the legislative committee had reviewed this request and actively supports the passage of such legislation. H.B. 2249 was therefore introduced and has passed the House of Representatives to be considered by the Arizona Senate in the near future.

IT WAS MOVED AND CARRIED TO SUPPORT LEGISLATION INTRODUCED TO AMEND THE MEDICAL PRACTICE ACT BY PROVIDING FOR A NEW DEFINITION OF ADVERTISING UNDER PROFESSIONAL CONDUCT TO READ "ADVERTISING A FALSE, DECEPTIVE OR MISLEADING MANNER, ADVERTISING THE QUALITY OF MEDICAL SERVICES, ADVERTISING BY IN-PERSON SOLICITATION AND ADVERTISING PROCEDURES AND PRACTICES WHICH ARE ILLEGAL."

AMA DELEGATION

LETTERS OF APPRECIATION

Letter was received from Tom E. Nesbitt, M.D., AMA President-Elect, expressing his personal appreciation for the Association's hospitality room. Letter was also received from David Beyer, student, University of Arizona thanking the Association and the Arizona Delegation for the opportunity to attend the AMA convention in Chicago.

OTHER BUSINESS

I.R.S. AUDIT

Mr. Robinson informed the Board of Directors that we had received notice from the I.R.S. that the Association would undergo an audit for the year December 31, 1975. This audit will take place sometime in late March. RECEIVED FOR INFORMATION.

AMSA MEMBERSHIP APPROVAL

IT WAS MOVED AND CARRIED TO APPROVE THE MEMBERSHIP OF THE FOLLOWING AMSA MEMBERS INTO THE ARIZONA MEDICAL ASSOCIATION:

	Class of
Gae Rodke	1981
Charlotte Richards	1981
John Donovan	1981
Paul Preston	1981
John Meyers	1981

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education.

Arizona Heart Institute
Arizona Medical Association, Inc.
Arizona State Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates
Phoenix Indian Medical Center
Phoenix Psychiatric Council
Maricopa County General Hospital, Phoenix
Memorial Hospital, Phoenix
St. Luke's Hospital & Medical Center, Phoenix
St. Joseph's Hospital & Medical Center, Phoenix
Tucson Hospitals Medical Education Program, Tucson
U of A College of Medicine
Veterans Administration Center, Prescott
Veterans Administration Hospital, Phoenix

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

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Individual program listings below indicate Category of CME. Where not indicated, no credit has been approved. For further information on any programs, contact person named.

JUNE

General Staff Quarterly Meeting

June 21, 1978. Chimney's Restaurant, Glendale, AZ. Sponsor: Glendale Samaritan Hospital. Contact: Richard Jaynes, M.D., Glendale Samaritan Hospital, 6010 West Northern, Glendale, AZ 85301. Approved for 1 hour of category 1 credit.

Medical History Society of Arizona

June 26, 1978. Contact: William C. Weese, M.D., (602) 265-0000.

Medical Horizons: Advances in Therapeutics
June 30-July 2, 1978, Little America Hotel, Flagstaff, AZ. Sponsor: College of Medicine, U. of A. Contact: Anthony F. Vuturo, M.D., Office of Continuing Medical Education, U of A, Tucson, AZ 85724. Approved for 10 hours of category 1 credit.

JULY

First Annual Summer Pediatric Conference Preparation for Recertification
July 7, 8, 9, 1978. 7:30 a.m.-2 p.m., Little America, Flagstaff, AZ. Sponsor: CME, U of A Health Sciences Center. Contact: Anthony F. Vuturo, M.D., ASHSC, 1501 N. Cmapbell Ave., Tucson, AZ 85724. Approved for 17.5 hours category 1 credit.

21st Annual Ruidoso Family Practice Seminar
July 17-20, 1978. Headquarters Inn, of the Mountain Gods at Mescalero, New Mexico. Sponsors: New Mexico Chapter of the American Academy of Family Physicians. Contact: Bob Reid, Convention Dir., P.O. Box 456, Sunland Park, N.M. 88063. Aapproved for category 1 credit.

A Day of Surgery
July 28, 1978. Little America Hotel, Flagstaff, AZ. Sponsor: College of Medicine, AZ Chapter of American College of Surgeons. Contact: C.H. Fredell, M.D., 120 W. Fine St., Flagstaff, AZ 86001. Approved for 7 hours of category 1 credit.

Coconino County Medical Society 14th Summer Medical Seminar
July 29 & 30, 1978. Little America, Flagstaff, AZ. Sponsor: Coconino County Medical Society. Contact: Paul Lundstrom, M.D., 120 W. Fine, Flagstaff, AZ 86001. Approved for 8 hours of category 1 credit.

SEPTEMBER

Arizona Society for Respiratory Therapy
September 6, 7, 8, Tucson, Arizona. Contact: Steve Krug, Chairman, c/o Tucson General Hospital, 3838 N. Campbell Ave., Tucson, AZ 85719

38th Annual AMA Congress On Occupational Health
September 14-16, 1978, Tucson, AZ. Contact: Barbara S. Jansson, Dept. of Environmental, Public & Occupational Health, AMA, 535 N. Dearborn St., Chicago, IL 60610. Approved for 15 hours of category 1 credit.

MONTHLY OR WEEKLY

Headache Rounds
Tuesday afternoons or Thursday mornings weekly. Sponsor: Headache Group of the Southwest. Contact: Peggy Voigt, 2610 W. Bethany Home Rd., Phoenix, AZ 85017, or phone 242-6646.

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m., J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8-10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., Arizona Medical Center, Dept. of Radiology, Tucson AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m. in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology, Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

Staff Education Conference
Wednesdays, weekly, 1 p.m., Arizona State Hospital, Phoenix, AZ. Sponsor: Arizona State Hospital. Contact: Howard E. Wulsin, M.D., Arizona State Hospital, 2500 E. Van Buren, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Road, Phoenix, AZ 85013. Sponsor: St. Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

Neurosurgery Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.

Neuro-ophthalmology Conference
Tuesdays, 11:30 a.m. in neuropathology conference room.

Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Room—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists.

Neuropathology of Gross Specimens Conference
Thursdays, 7:30-8:30 a.m. in the Morgue.

Neurology-Neurosurgical Clinical-Neuroradiological and Neuropathological Conference
Fridays, 8-9 a.m., First Floor Conference Room.

Neuropathology or Neuroradiology Conferences
Fridays, 9 a.m., Neuropathology in Neuropathology Conference Room, Neuroradiology in First Floor Conference Room.

Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Room.

Neurosurgical Journal Club
Saturdays, 9-11 a.m., in Eighth Floor Conference Room.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.

Patient Staffing Conference
Three times weekly.

DESERT SAMARITAN HOSPITAL

Wednesday evenings, 7 p.m. Sponsor: Desert Samaritan Hospital. Contact: L.A. Rosati, M.D., Laboratory, Desert Samaritan Hospital, Mesa, AZ 85202. Approved for category 1 credit.

GOOD SAMARITAN HOSPITAL

Clinical Cancer Conference
3rd Wednesday, every month, Butler Bldg. Conference Room, Good Samaritan Hospital. Contact: John A. Bruner, M.D., 926 E. McDowell Road, Phoenix, AZ 85006. Approved for 1 hour per session category 1 credit.

GOOD SAMARITAN HOSPITAL

Pulmonary Disease Grand Rounds
Mondays, 12 Noon, D-5 North Conference Room, Good Samaritan Hospital, Phoenix, AZ. Sponsor: Pulmonary Disease Teaching Service, Good Samaritan Hospital. Contact: Bernard E. Levine, M.D., Pulmonary Function Laboratory, Good Samaritan Hospital, 1033 E. McDowell Road, Phoenix, AZ 85006. Approved for 1 hour category 1 credit.

MARYVALE SAMARITAN HOSPITAL

Continuing Medical Education Program
Maryvale Samaritan Hospital, every other Wednesday, 7-8 a.m., January 25, 1978 through December 27, 1978. Sponsor: Medical Staff of Maryvale Hospital. Contact: Richard C. Christensen, M.D. Approved for 1 hour per session category 1 credit.

MEMORIAL HOSPITAL

1201 S. 7th Ave., Phoenix, AZ 85036. Sponsor: Memorial Hospital. Approved for category 1 credit.

Monthly Medical Education Seminar
Third Monday, 6:30 p.m., Kiva Conference Room. Contact: George Scharf, M.D.

Tumor Board Conference
Every Friday at Noon, Kiva Conference Room. Contact: Howard Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 E. 4th St., Scottsdale, AZ 85251. Sponsor: Scottsdale Memorial Hospital. Contact: W.S. Williams, M.D., Director of Medical Education.

Clinical Pathological Conference
4th Monday, monthly, 12:45 p.m.

Family Practice Conference
1st Monday, monthly, 12:45 p.m.

Medical Grand Rounds
3rd Monday, monthly, 12:45 p.m.

Morbidity & Mortality Conference
2nd Monday, monthly, 12:45 p.m.

ST. MARY'S HOSPITAL & HEALTH CENTER

1601 West St. Mary's Rd., Tucson, AZ 85703. Sponsor: St. Mary's Hospital & Health Center. Contact: see below.

Monthly Specialty Conference of the Department of Surgery
First Monday, monthly, 7:30 a.m., Century Room A. Contact: Medical Staff Office. Approved for 1 hour of category 1 credit.

Grand Rounds: Medical, Surgical, Family Practice, Pathology, Radiology
Weekly, Thursday, 7-8:30 a.m. Contact: Richard A. Silver, M.D. Approved for 1 hour category 1 credit.

U OF A HEALTH SCIENCES CENTER

Tucson, AZ 85724. Sponsor: U of A College of Medicine. Contact: See below. Approved for category 1 credit.

Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 p.m. Contact: John Boyer, M.D., Dept. of Internal Medicine.

Cerebrovascular Disease Conference
Mondays, 5-6 p.m., weekly, Room 5505. Contact: Wesley Moore, M.D., Depts. of Surgery, Neurology & Radiology.

Dean's Clinical Rounds
July 19, 1978, Main Auditorium, UAHSC. Contact: Louis J. Kettel, M.D.

Endocrinology Seminar
Every Thursday, Noon-1 p.m., 1st, 3rd & 5th Thursday, Room N318, VA Hospital. 2nd & 4th Thursday, Room 6505, Tucson Medical Center. Contact: Dept. of Internal Medicine.

Hematology-Oncology Clinical Conference
Every Tuesday, Noon-1 p.m., 1st, 3rd & 5th Tuesdays, Room 6505, Tucson Medical Center. 2nd & 4th Tuesdays, Room N318, VA Hospital. Contact: Sidney Salmon, M.D., Dept. of Internal Medicine.

Medical Grand Rounds
Every Wed., Noon-1 p.m., 1st, 3rd & 5th Wed., Staff Conf. Room, VA Hospital. 2nd & 4th Wed., Room 5403, UAHSC. Contact: Jay Smith, M.D., Dept of Internal Medicine.

Nuclear Medicine
Thursday, 7:30 a.m., Radiology Conf. Room, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept of Diagnostic Radiology.

Neurology Grand Rounds
Every Friday, Noon, 1st, 3rd & 5th Fridays, UAHSC, Room 7505. 2nd & 4th Fridays, VA Hospital. Contact: William A. Sibley, M.D., Dept. of Neurology.

Neuropathology Review Conference
Thursdays, 8:30 a.m., Room 5120, UAHSC. Contact: Peter C. Johnson, M.D.

Neurosciences Seminar
Every Friday, 7:30 a.m., UAHSC, Room 7505. Contact: Colin R. Bamford, M.D., or Jose F. Laguna, M.D., Dept. of Neurology.

Pathology Seminar
Friday, 4:30-5:30 p.m., Room 5120, UAHSC. Contact: Kenneth Ryan, M.D., Dept. of Pathology.

Tucson Pathologist Conference
First Monday of the month, 7:30-9 p.m. Rotation between Tucson Pathology Conf Rooms. Contact: Merel Clard, M.D., Tucson Pathologists.

Pediatric Grand Rounds
Every Tuesday, 12:30-1:30 p.m. Alternates between Tucson Medical Center & UAHSC. Contact: Hugh Thompson, M.D., Dept. of Pediatrics.

Problem Case Workshop
3rd Monday of each month, 7:30 a.m., Room 4410, UAHSC. Contact: H.E. Cross, M.D., Ph.D., Div. of Ophthalmology.



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Psychiatric Grand Rounds
Every Wednesday, Sept. to May 4, 5:30 p.m., Room 8403 UAHSC. Contact: Alan Levenson, M.D., Dept. of Psychiatry.

Chest Radiology
Monday, 5-6 p.m., weekly, Room 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Gastrointestinal Radiology
Mondays, 7:30 a.m., weekly except for summer months. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Radiation Oncology Planning Conference
Friday, weekly, 8:30-10 a.m., UAHSC, Room 0655. Contact: Robert S. Heusinkveld, M.D., Radiation Oncology Section.

Radiology-Rheumatology Conference
Thursday, 8 a.m., UAHSC, Library Room 1535C. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Vascular Radiology
January-June 1978, 7:30 -8:30 a.m., every Friday. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Radiology Interesting Case Conference
Thursday, Noon, VA Hospital. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Room. Contact: Irwin M. Freundlich, M.D., Dept of Radiology.

Trauma Conference
Every Thursday, 4-5 p.m. UAHSC, Room 5505. Contact: Martin Silverstein, M.D., Dept. of Surgery.

Grand Ward Rounds—Trauma
Every Tuesday, 8 a.m., UAHSC. Contact: Martin Silverstein, Surgery Dept., Trauma Section.

Monthly Meeting Tucson Ultrasonography Group
First Wednesday of month, 4:30-5:30 p.m., UAHSC. Contact: Irwin M. Freundlich, M.D.

Tucson Urological Society Scientific Meeting
Monthly. Contact: George W. Drach, M.D., Depts of Surgery & Urology.

General Urology Conference
Tuesday, 5-6 p.m. Thursday, Noon-1:30 p.m., UAHSC Meeting Rooms & VA Hospital. Contact: George W. Drach, M.D., Dept. of Surgery/Urology Section.

VETERANS ADMINISTRATION HOSPITAL

7th Street and Indian School Rd., Phoenix, AZ 85012. Sponsor: VA Hospital. Contact: Jasper L. McPhail, M.D. Approved for category 1 credit.

Cancer Symposium
Each Wednesday, 1 p.m., T-5.

Cardiology Conference
2nd Thursday, 1 p.m., T-5.

Medical-Surgical Chest Conference
1st and 3rd Thursday, 1 p.m., T-5.

Clinicopathology Conference
4th Thursday of 3rd Mo. (Mar., June, Sept. & Dec.), 1 p.m., T-5.

Dermatology Conference
1st, 2nd & 3rd Wednesday.

Hepathology Conference
2nd and 4th Tuesday, 1 p.m., 2128.

Gastroenterology Conference
1st and 3rd Tuesday, 1 p.m., T-5.

GI Conference
(Special Program with U of A Consultants)
4th Friday, 1 p.m., T-5.

GI Radiology Clinical Correlation Conference
Each Tuesday, 1 p.m., T-5.

Medical Surgical GI Conference
1st & 3rd Friday, 2 p.m., 2128.

Medical Service Grand Rounds
Each Friday, 11 a.m., T-5.

Surgery-Pathology Conference
Each Thursday, 7 a.m., 2128.

Urology-Pathology Conference
Each Wednesday, 7 a.m., 2128.

Brief Summary of Prescribing Information
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Capsules and Oral Solution
For complete information, consult Official Package Circular. (12) TEGOPEN 9/11/

Indications: Although the principal indication for cloxacillin sodium is in the treatment of infections due penicillinase-producing staphylococci, it may be used initiate therapy in such patients in whom a staphylococc infection is suspected. (See Important Note below.)

Bacteriologic studies to determine the causative organisms and their sensitivity to cloxacillin sodium should performed.

Important Note: When it is judged necessary that treatment be initiated before definitive culture and sensitiv results are known, the choice of cloxacillin sodium shou take into consideration the fact that it has been shown be effective only in the treatment of infections caused pneumococci, Group A beta-hemolytic streptococci, a penicillin G-resistant and penicillin G-sensitive staphlococci. If the bacteriology report later indicates t infection is due to an organism other than a penicil G-resistant staphylococcus sensitive to cloxacillin sodiu the physician is advised to continue therapy with a dr other than cloxacillin sodium or any other penicillina resistant semi-synthetic penicillin.

Recent studies have reported that the percentage staphylococcal isolates resistant to penicillin G outsi the hospital is increasing, approximating the high pe centage of resistant staphylococcal isolates found in t hospital. For this reason, it is recommended that a pe cillinase-resistant penicillin be used as initial therapy f any suspected staphylococcal infection until culture a sensitivity results are known.

Cloxacillin sodium is a compound that acts through mechanism similar to that of methicillin against penicil G-resistant staphylococci. Strains of staphylococci res tant to methicillin have existed in nature and it is kno that the number of these strains reported has been increa ing. Such strains of staphylococci have been capable producing serious disease, in some instances resulting fatality. Because of this, there is concern that widespre use of the penicillinase-resistant penicillins may result the appearance of an increasing number of staphylococc strains which are resistant to these penicillins.

Methicillin-resistant strains are almost always resista to all other penicillinase-resistant penicillins (cros resistance with cephalosporin derivatives also occu frequently). Resistance to any penicillinase-resistant pe cillin should be interpreted as evidence of clinical res tance to all, in spite of the fact that minor variations *in vitro* sensitivity may be encountered when more tha one penicillinase-resistant penicillin is tested against t same strain of staphylococcus.

Contraindications: A history of a previous hypersen tivity reaction to any of the penicillins is a contraindicatio

Warning: Serious and occasionally fatal hypersensitivi (anaphylactoid) reactions have been reported in patien on penicillin therapy. Although anaphylaxis is more fr quent following parenteral therapy it has occurred patients on oral penicillins. These reactions are more a to occur in individuals with a history of sensitivity multiple allergens.

There have been well documented reports of individua with a history of penicillin hypersensitivity reactions wh have experienced severe hypersensitivity reactions whe treated with a cephalosporin. Before therapy with a pen cillin, careful inquiry should be made concerning previo hypersensitivity reactions to penicillins, cephalosporin and other allergens. If an allergic reaction occurs, the dru should be discontinued and the patient treated with t usual agents, e.g., pressor amines, antihistamines, an corticosteroids.

Safety for use in pregnancy has not been establishe

Precautions: The possibility of the occurrence of supe infections with mycotic organisms or other pathoge should be kept in mind when using this compound, as wi other antibiotics. If superinfection occurs during therap appropriate measures should be taken.

As with any potent drug, periodic assessment of orga system function, including renal, hepatic, and hemat poietic, should be made during long-term therapy.

Adverse Reactions: Gastrointestinal disturbances, suc as nausea, epigastric discomfort, flatulence, and loos stools, have been noted by some patients. Mildly elevate SGOT levels (less than 100 units) have been reported in few patients for whom pretherapeutic determinations we not made. Skin rashes and allergic symptoms, includir wheezing and sneezing, have occasionally been encou tered. Eosinophilia, with or without overt allergic ma festations, has been noted in some patients during therap

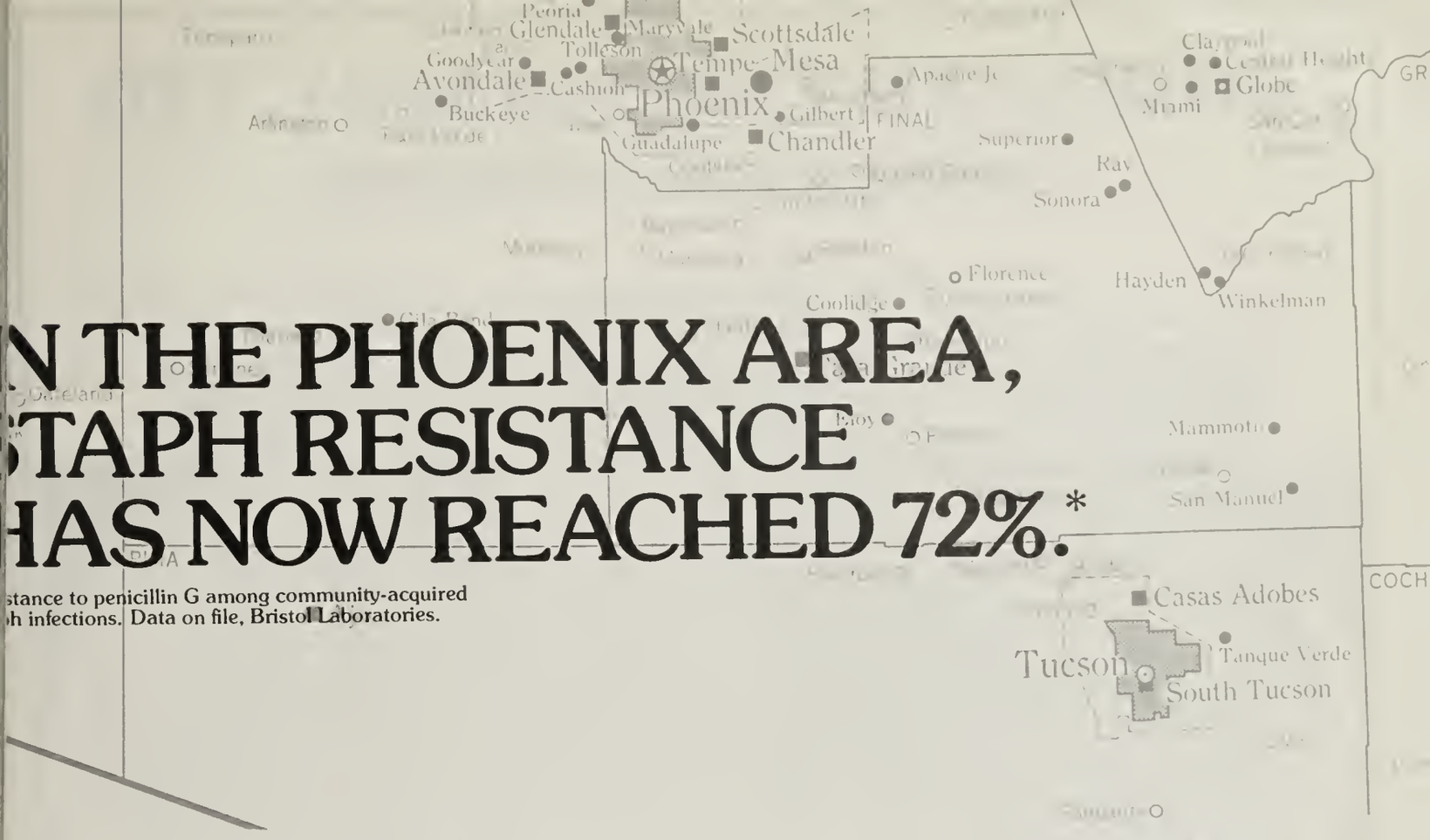
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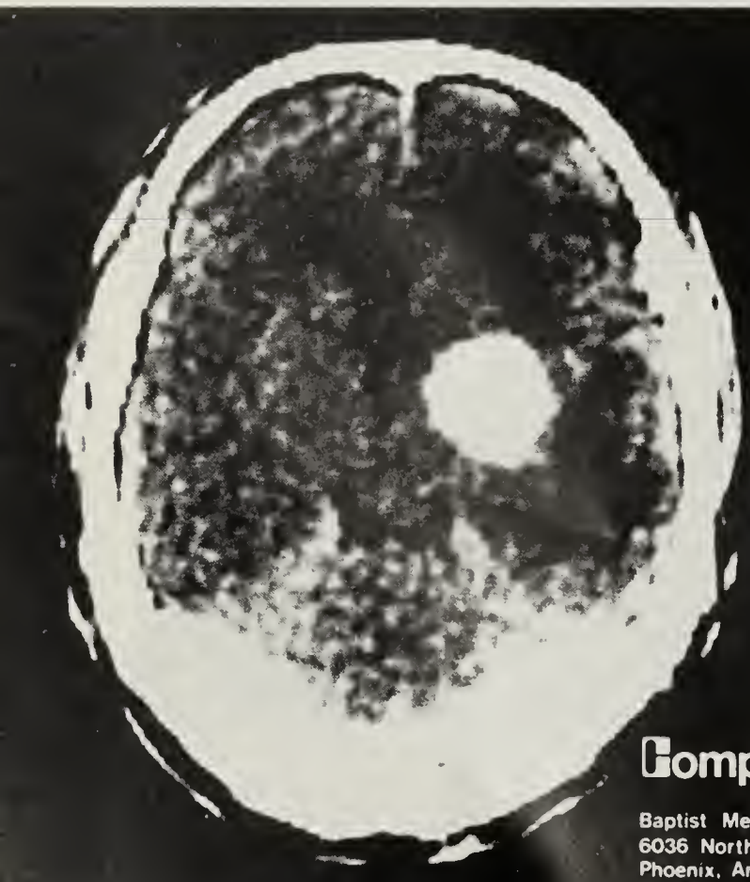
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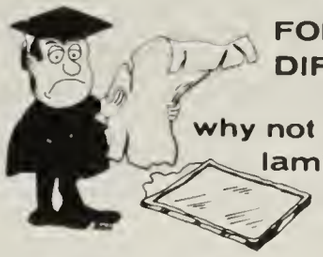
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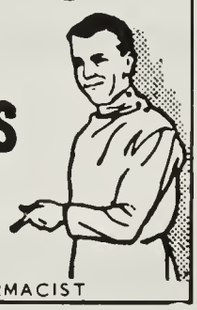
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